THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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Various circumstances indicate that grape degeneration as it occurs in Florida may be due to the Pierce's disease virus, according to Warren N. Stoner, Loren H. Stover, and G. K. Parris, page 341.

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FIELD AND LABORATORY INVESTIGATIONS INDICATE GRAPE DEGENERATION IN FLORIDA IS DUE TO PIERCE'S DISEASE VIRUS INFECTION

Warren N. Stoner, Loren H. Stover, and G. K. Parris [1]

Wild native grapes are found growing profusely in woodlands in Florida. The early Spanish settlers in the 16th century noted this and made probably the first efforts to establish vineyards of the European grapes, Vitis vinifera, in the State. These plantings failed completely, and all succeeding attempts up to the present time to grow grapes of this species have failed.

In 1891 De Pass planted 60 varieties of grapes near Lake City, Florida. Three years later this planting was dying and being replanted. About this same time the varieties Concord, Ives, and Niagara, of the species Vitis labrusca, were planted near Orlando, Florida. They too soon failed.

By 1894 there was a fairly large grape acreage in the State (500 acres in Orange County alone), but within six to eight years these plantings were all dead. The heavy losses suffered by the growers were brought to the attention of the Federal Government and in 1899 a comprehensive grape test was established at Earlton, Florida. This planting also failed, but it did give some indication that certain rootstocks, and the Texas hybrids developed by Munson, were longer lived in Florida.

It should be noted that the period 1891-1894 in Florida parallels the period 1884-1900 when what we now know as Pierce's disease was destroying thousands of acres of grapes in California (4).

In 1924 Hawkins of Eustis, Florida planted nearly 100 varieties of Vitis vinifera on land known to be free of the grape Phylloxera. The planting bore almost no fruit and was abandoned. By this time, however, Florida plantings of the Texas hybrids of Munson were quite common. The varieties Extra (Florida Beacon), Carman, R. W. Munson, Muench, and Bailey were all showing promise and carload lots of grapes were being shipped from Orlando to northern markets.

Rhoads (7) in his description of root rot, chlorosis, and irregular water relations includes symptoms similar to those of what has been since proven to be Pierce's disease of grape in California. By inference these symptoms, taken as a whole, present a good general description of the syndrome of Grape Degeneration in Florida. To be sure, root rot, chlorosis, and high water may have played a part in debilitating grapes in Florida, but fertilization and minor element studies have shown that none of the fertilizer programs used to date will alleviate grape degeneration (6). Soil fumigation practiced where degenerate vines have been pulled has failed to protect replants (6), and degeneration kills the entire vineyards regardless of the location of the vines, even on well drained slopes. Figure 66 of Rhoads' bulletin (7) illustrates a vineyard supposedly being depleted by Clitocybe tabescens root rot. (It is still unknown whether most of the degenerate vines in the past were actually killed by root-invading fungi or whether these organisms were secondary in nature. No visible organism has been consistently associated with degeneration in Florida (6)). Examination of this illustration shows that native weeds and grasses are invading the vineyards where the vines are dying, and the general appearance is similar to that of a Pierce's disease-affected vineyard in California. Chlorosis as illustrated in Figure 73 of Rhoad's bulletin (7), resembles several illustrations of Pierce's disease given by Hewitt et al. (4).

The grape industry decreased in Florida during the thirties and little replanting was done in the now declining vineyards. In spite of the discouraging past, K. W. Loucks at the Watermelon and Grape Investigation Laboratory at Leesburg persisted in the search for grapes that would grow in Florida. An unpublished report written by Loucks about 1937 shows that well over 100 varieties were under test, on their own roots and grafted to several types of rootstocks. Some of these showed a little promise but the overall results were dead and dying vines.

The work was continued, however, at Leesburg from 1944 by Loren H. Stover, and from 1945 to the present by Stover and Parris. By 1946 it was believed that such varieties as Concord, Niagara, Portland, and Fredonia, that failed on their own roots, could be grown if grafted to rootstock varieties such as Extra (Florida Beacon), R. W. Munson, and Black Spanish. In 1948 Dickey, Stover, and Parris (2) recommended the following varieties for Florida: Extra (Florida Beacon), Muench, and Bailey on their own roots; Fredonia, Carman, and Niagara grafted on a rootstock (see the preceding paragraph). Today these recommendations are obsolete, and Parris

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(6) has said "Our grafting tests are doing none too well and from the reactions of the grafted plants we know that grafting is not the answer [to degeneration]. We therefore have no grape variety or combination of varieties in a grafted plant which we can recommend for planting in Florida at the present time (1951)."

Parris (6) reviewed the grape situation in Florida at the Annual Meeting of the State Plant Pathologists, April 1951. His descriptions of grape degeneration (cause unknown) and its behavior in the field indicated to the senior author that the condition was very similar to Pierce's disease of grape as described by Hewitt et al. (4). The following observations were made at the farm of the Watermelon and Grape Investigations Laboratory at Leesburg, and in other locations noted, on the 5th, 6th, and 19th of June, 1951.

Several varieties of different ages were observed and the following is a composite description of the symptoms of grape degeneration in Florida. All varieties did not show all of the symptoms. In most instances declining plants show an interveinal chlorosis of the leaves in varying degree (Fig. 1A). When the mottling is well developed a glossy appearance is imparted to the dorsal surface of the yellowed portion, and the leaves are stiffened in texture. Marginal burning of the basal leaves of the canes was quite evident (Fig. 1B), and in some instances individual canes were badly scorched. In cases where the leaf burning had progressed considerably, the leaf lamina abscissed from the petiole, but the petiole did not absciss from the stem (Fig. 2)

The junior authors have observed that vines affected with degeneration are slow to start in the spring. In the case of vines known to have the condition in previous years, asymmetrical growth is quite evident now; that is, one arm fails to produce growth or has weak suckers at its base. The other arm grows fairly normally but is later than the majority of the other vines (Fig. 3). The growth of the affected plants becomes progressively less each year and death ultimately follows depending on the variety and individual plant. Canes of vines suffering with degeneration are dwarfed and the length of the internodes is shortened.

Occasionally vines with enough vigor to produce fruit have clusters that are flaccid and withered. Some varieties stop producing fruit as the disease progresses.

In the fall, canes of degenerate plants fail to mature properly and green areas persist between the nodes.

Stover and others in the Leesburg area often have difficulty rooting cuttings in the nursery. Many cuttings do not grow at all and others grow but weakly the first season or two, show degeneration symptoms and then die.

Past experience in commercial and experimental plantings has made it possible to group grapes in Florida into six classes according to longevity. This grouping may vary depending on rootstock but in general the relationship holds good as follows: Group I, Vitis vinifera, shortest lived, about two years or less (over 100 varieties tried). Group II, Vitis labrusca, varieties in this group such as Concord, Niagara, Worden, Fredonia and Portland live slightly longer, three to four years. Group III contains only the variety Catawba which will live slightly longer than the varieties in Group II, three to five years. Group IV contains the Texas Post Oak Hybrid varieties Extra (Florida Beacon), Carman, Bailey, and Muench which will live five to eight years. Hybrids of many varieties x Vitis simpsoni are now under trial at Leesburg and are living well since 1946 with no sign of degeneration. These have been placed in Group V. The native wild grape Vitis simpsoni thrives and gives no indication of degeneration, and this species comprises Group VI.

It is possible to extend the life span of some grape varieties slightly in Florida by grafting to certain rootstocks, but in no case has this extension been of sufficient value for commercial production.

Specimens of degenerate vines were collected from the field in the Leesburg area on June 5 and 6, 1951. Fresh cross sections were made of these specimens in the laboratory and microscopically examined. The xylem elements of the canes and the petioles of the leaves contained gum deposits and tyloses. Esau (3) has shown that gum deposition and tylosis formation in the xylem elements of grapes can be induced by infection with Pierce's disease virus.

It has long been known in central Florida that when Bermuda grass, Cynodon dactylon, moves into a vineyard the grapes go out due to degeneration. For this reason insect collections were made on the fifth of June, 1951, by sweeping this grass at Leesburg where degeneration had almost destroyed a seven-year old commercial vineyard of the variety Extra. Large numbers of the yellow-headed sharpshooter, Carneocephala flaviceps (Riley) [2] were taken. This insect is closely related to Carneocephala fulgida Nott., one of the principal vectors of Pierce's disease in

[2]The authors are indebted to Professor Dwight M. DeLong, Ohio State University for determination of the leafhopper species listed.

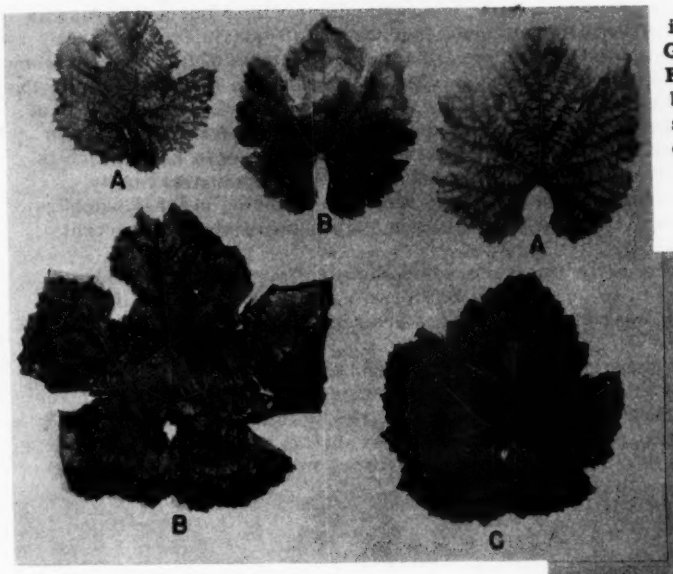

FIGURE 1. Leaves A show interveinal chlorosis typical of Grape Degeneration as seen in Florida. Leaves B show marginal burning which increases as the season progresses. C is healthy check leaf. All x approximately 1/4.

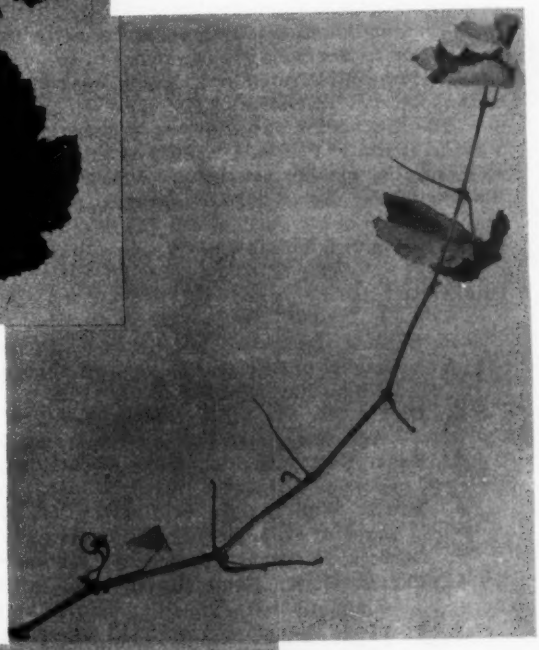

FIGURE 2. Single grape cane affected with Degeneration showing severely scorched terminal leaves. Note the lower leaf laminae have abscissed from the petioles but the petioles have not abscissed from the cane. x approximately 3/16.

FIGURE 3. A young grape plant showing failure of left arm to grow. Growth on right arm is much delayed and weak. Compare to plant in background. Note thick ground cover of Bermuda grass.

California, listed by Hewitt et al. (4), Winkler (9), DeLong and Severin (1), and Severin (8). Both of these species favor Bermuda grass as a host plant. Winkler (9) lists Bermuda grass as being susceptible to Pierce's disease virus in California, and Osborn (5) states that Bermuda grass is one of the favored food plants for C. flaviceps. Further collections of C. flaviceps were made on June 6, 1951 at the Bream Lake Grape Nursery (Experimental farm Leesburg) where the yellow-headed sharpshooter was taken on grape as well as Bermuda grass. A few specimens of Draeculacephala portola Ball, and D. inscripta Van Duzee were collected by sweeping grasses near the shore of Bream Lake. Both of these species are related to D. minerva Ball, which is another of the principal vectors of Pierce's disease in California. Degeneration was quite evident in grapes of all ages at this location. C. flaviceps was also collected in large numbers at Eustis, Florida on Bermuda grass. Vineyards in this area were showing about 40 percent degenerate plants.

Examination of the flora at the collection locations noted show that many species of plants, in addition to Bermuda grass, known to be susceptible to Pierce's disease virus in California are present. Cultivated and wild legumes including clovers are growing adjacent to the vineyards suffering from degeneration.

The preceeding observations of grape degeneration in Florida are generally consistent with what is known of Pierce's disease of grape in California. Greenhouse tests are now being made to determine whether grape degeneration in Florida is being caused by infection with Pierce's disease virus. The foregoing observations indicate strongly that such is the case. It is possible that a portion of the running-out and degeneration troubles of grapes in the southeastern United States is due to infection with Pierce's disease virus.

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ADDITIONAL INSECT VECTORS OF WESTERN X-DISEASE [1]

E. W. Anthon [2] and H. R. Wolfe [3]

The first insect vector discovered for Western X-disease of peaches was the geminate leafhopper, Colladonus geminatus (Van D.) (Wolfe, Anthon and Jones, 1950).

During 1949-1950 the authors demonstrated successful transmissions of Western X-disease to peach varieties in the field by the leafhoppers, Fieberiella florii (Stal), Keonolla confluens (Uhl), and Scaphytopius acutus (Say) [4]. In greenhouse tests, successful transmissions of Western X-disease were made by F. florii and K. confluens to Lovell peach seedlings.

In the Pacific Northwest the favorite host plant of F. florii is sweet cherry, although it has been found on chokecherry. K. confluens favors willow, which is especially common along the irrigation ditch banks in the Washington fruit growing areas. This leafhopper has also been

Table 1. Transmission of Western X-disease to peach by Fieberiella florii, Keonolla confluens, and Scaphytopius acutus, Wenatchee, Washington, 1948-1949.

Species and stage of insects	Number tests	Kind of inoculum	Field or greenhouse tests	Days insects on inoculum	Trans-missions	Negative tests
F. florii nymphs	1	Cherry	Field	13	1	
F. florii nymphs	3	Peach	Field	13		3
F. florii nymphs and adults	3	Peach	Field	3-13	1	2
F. florii adults	1	Peach	Field	10		1
F. florii nymphs	1	Cherry	Greenhouse	Developed on inoculum		1
F. florii adults	3	Cherry	Greenhouse	Developed on inoculum	1	2
F. florii nymphs	5	Peach	Greenhouse	5		5
Sub-total	17				3	14
S. acutus nymphs	2	Peach	Field	4		2
S. acutus nymphs and adults	2	Peach	Field	4-7	1	1
S. acutus adults	9	Peach	Field	4-12		9
Sub-total	13				1	12
K. confluens nymphs	46	Peach	Field	2-15	1	45
K. confluens adults	9	Peach	Field	3-14		9
K. confluens nymphs	1	Peach	Greenhouse	8		1
K. confluens nymphs and adults	2	Peach	Greenhouse	8-10		2
K. confluens adults	15	Peach	Greenhouse	33-41	2	13
Sub-total	73				3	70
Grand Totals	103				7	96

[1] Scientific Paper No. 1013, Washington Agricultural Experiment Stations, Institute of Agricultural Sciences, The State College of Washington, Pullman.

[2] Assistant Entomologist, Tree Fruit Experiment Station, Wenatchee, Washington. Supported in part by the United States Department of Agriculture, Bureau of Entomology and Plant Quarantine, Division of Fruit Insect Investigation.

[3] Assistant Entomologist, Tree Fruit Experiment Station, Wenatchee, Washington.

[4] Leafhoppers determined by David A. Young, U. S. Department of Agriculture, Division of Insect Identification.

collected from alfalfa and Lombardy poplar. In late summer it occasionally becomes abundant on peach and cherry. The primary host of S. acutus is alfalfa, although it is often found on strawberry and has also been collected from chokecherry. These insects are able to survive for long periods on stone fruits and have therefore been the subject of vector research.

Insects used in vector experiments were collected in the field. Some of the leafhoppers in this series of tests were fed on Western X-affected peach and some on Western X-little cherry (sweet cherry) before transfer to healthy peach. Test trees have included both standard peach varieties and potted Lovell peach seedlings.

Our first evidence of insect transmission of Western X-disease from Western X-little cherry infected sweet cherry to peach was secured in the late summer of 1950, at Wenatchee. The insect vector was the leafhopper, F. florii. In the field plot experiments the insects were collected from cherry trees as nymphs and fed on Western X-little cherry inoculum. Adult insects used for transmission in the greenhouse were collected from diseased cherry in the field and transferred to healthy potted test trees. With each test tree a check tree was provided and all check trees remained healthy, both in the greenhouse and in the field trials.

In experiments conducted in 1949 and 1950 at Wenatchee, involving transfers of F. florii, K. confluens, and S. acutus, there were 103 tests, with 7 transmissions. The number of leafhoppers used in each transfer varied from 1 to 50. The insect transmissions were conducted in 1949, but test trees did not show Western X-disease symptoms until 1950. Investigations thus far have not revealed definite information concerning the latent period of the virus in the insect. Details of the experiments are summarized in Table 1.

Although experimental insect transmission of Western X-disease has now been obtained with these cicadellids, the part they play in the natural spread of the disease is not known.

SUMMARY: -- The Western X-disease of peaches has been transmitted from peach to peach by Fieberiella florii, Keonolla confluens, and Scaphytopius acutus in experiments at Wenatchee, Washington.

F. florii also transmitted Western X-disease to peach from sweet cherry affected with Western X-little cherry under both field and greenhouse conditions.

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TRANSMISSION OF WESTERN X-DISEASE VIRUS FROM CHOKECHERRY TO PEACH BY COLLADONUS GEMINATUS (VAN D.)[1]

George H. Kaloostian

The transmission of Western X-disease virus or viruses from infected peach to healthy peach by the geminate leafhopper, Colladonus geminatus (Van D.), has been demonstrated repeatedly in Oregon, Washington, and Utah. (1, 2, 3). In January and February 1951, at Logan, Utah, two cases of Western X-disease developed in potted Lovell seedling peach trees after inoculation by geminate leafhoppers that had been fed on infected chokecherry, Prunus virginiana var. demissa (Nutt.). The retention of the virus by the insects for at least 47 days is indicated. The procedures employed in these tests are as follows:

Case No. 1: A complex of leafhoppers consisting of C. geminatus and Aceratagallia sp. was collected from alfalfa in August 1950, and then bagged for 30 days on chokecherry infected with Western X-disease. After this inoculum feeding period, 40 adults including both species were transferred to a healthy, field-plot peach tree, where they were confined for 28 days. The tree was still dormant when this report was written.

Fifteen surviving adults were transferred from the field-test tree to a healthy potted Lovell seedling in the greenhouse, where they were allowed to feed for 19 days. This tree died in January in 1951 without showing symptoms of Western X-disease.

On November 13, 1950, two surviving adults of C. geminatus were transferred to another potted Lovell seedling in the greenhouse. They remained alive on this tree until December 4, a period of 21 days. Symptoms of Western X disease were apparent in this tree on January 23, 1951.

Case No. 2: Leafhoppers were collected from alfalfa in August 1950, and bagged on diseased chokecherries for a feeding period of 32 days, to permit the acquisition of the virus. At the end of this period, a complex of 50 adults and nymphs of C. geminatus and Aceratagallia sanguinolenta (Prov.) was transferred to a healthy field-plot peach tree for a feeding period of 29 days. A mixture of 16 adults and nymphs of these two species was then transferred to a potted Lovell peach seedling in the greenhouse, on which they were allowed to feed for 18 days. No symptoms had appeared on the latter tree by March 1, 1951, and the field tree was still dormant.

On November 11, 1950, the leafhoppers were separated as to species and one, three, and five C. geminatus, respectively, were transferred to three potted, healthy Lovell peach seedlings. After a test feeding period of 24 days, the insects were transferred to other healthy trees. The test tree that received the three C. geminatus developed symptoms of Western X-disease in February 1951. Neither of the other trees has shown symptoms to date, and the control trees are healthy.

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[1]This work was done in cooperation with the Utah Agricultural Experiment Station and U. S. Bureau of Plant Industry, Soils, and Agricultural Engineering, with funds authorized under the Research and Marketing Act of 1946.

TRANSMISSION OF WESTERN X-LITTLE CHERRY VIRUS FROM SOUR AND SWEET CHERRY TO PEACH BY COLLADONUS GEMINATUS (VAN D.) [1]

George H. Kaloostian

Transmission of the Western X-disease from infected peach to healthy peach by the geminate leafhopper (Colladonus geminatus (Van D.)) has been demonstrated in Oregon, Washington, and Utah (1, 2, 3). In February 1951, at Logan, Utah, the same species of leafhopper produced three cases of Western X-disease in potted Lovell peach seedlings after feeding on sour and sweet cherry trees infected with Western X-little cherry virus. The retention of the virus by the insects for at least 72 days is indicated. The procedures followed in the successful tests are described below.

A complex of leafhoppers, including C. geminatus and Aceratagallia sanguinolenta (Prov.), was collected from alfalfa in September 1950, and then bagged for 28 days on a sour cherry tree infected with Western X-little cherry disease. At the end of this infection feeding period 50 adults of these two species were transferred to a healthy peach tree for 33 days. This tree, which was growing in a field plot, was still dormant when this report was written so that the results of the test are not yet available.

Twenty-one surviving adults of both species were transferred from the test tree to a second healthy test tree, in this case a potted Lovell seedling in the greenhouse, where they were allowed to feed for 39 days. No symptoms of disease had appeared on this tree by March 1, 1951.

On December 7, 1950, all the A. sanguinolenta leafhoppers were dead, and those of C. geminatus that survived were transferred to seven potted, healthy Lovell peach seedlings. The test feeding periods were 24 days. Two of these seedlings developed symptoms of Western X-disease in February 1951. The number of leafhoppers involved in the successful tests was one and three adults per test tree respectively. None of the controls developed symptoms of Western X-disease.

In the third case of transmission, the leafhoppers were collected in August 1950, and bagged for one month on a sweet cherry tree infected with Western X-little cherry. Forty adults and nymphs were transferred to a healthy peach tree in the field plot, where they remained for a feeding period of 32 days. This tree is now dormant and the results of the test will not be known until the summer of 1951. Seven surviving adults were transferred to a one-month-old Lovell seedling in the greenhouse; the leafhoppers remained on this plant for 38 days. This tree appears to be healthy to date.

Three surviving C. geminatus adults from this test tree were transferred individually to one-week-old Lovell peach seedlings, where they remained until they were dead. One of these test trees showed symptoms of Western X-disease approximately two months after the completion of the 26-day transmission feeding period. The other test and control seedlings remained healthy.

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[1] In cooperation with the Utah Agricultural Experiment Station and U. S. Bureau of Plant Industry, Soils, and Agricultural Engineering. The work here reported was financed with funds authorized under the Research and Marketing Act of 1946.

TRANSMISSION OF WILT AND DECLINE DISEASE OF CHERRIES IN UTAH BY THE BLACK CHERRY APHID

George H. Kaloostian, Mervin W. Nielson, and L. S. Jones [1]

During the last two years the black cherry aphid (Myzus cerasi (F.)) has been used in tests of suspected vectors of the Western X virus in Utah. Attention has been given to both the Western X-little cherry disease on sweet and sour varieties on mazzard rootstocks and the Western X-wilt and decline disease as it occurs on cherries on mahaleb roots. Both disorders have been considered to be caused by the same virus or viruses that produce the Western X-disease in peach and chokecherry. Richards et al. (3, 4) have demonstrated in Utah that these disorders are caused by a virus or viruses that are bud- or graft-transmissible between peaches, chokecherries, and cherries in all combinations, except that they were never induced in cherries when infected peach buds were grafted directly into cherries, or when buds or grafts taken from wilting and declining cherry trees were placed on healthy cherry trees.

The concept of aphid transmission of the Western X group of diseases in stone-fruit trees is somewhat opposed to the general opinion that leafhoppers are the natural vectors. It has already been demonstrated in Oregon, Washington, and Utah that Colladonus geminatus (Van D.) is a vector of the virus from peach to peach (5, 6, 7), and recent investigations have demonstrated its transmission from cherry and chokecherry to peach (1, 2). In view of this evidence it is possible that the Western X-wilt and decline disease of cherries in Utah may be caused by a virus that is distinct from the one that causes Western X-disease of peach or Western X-little cherry in cherries on mazzard rootstocks.

Evidence of the transmission of Western X-wilt and decline virus by the black cherry aphid was first obtained in Utah in 1949. Two out of five test trees fed upon by aphids taken directly from trees infected with Western X-little cherry wilted and died. The other three test trees and five check trees remained healthy. The check trees had not been exposed to the feeding of non-infective aphids. Previous experience had shown that the numbers of aphids that were used should not be expected to have a lethal effect from their toxins alone.

In 1950 a large-scale experiment was conducted in two field plots, one at Farmington and the other near Logan, Utah. Napoleon sweet cherry trees on mahaleb rootstocks were alternated with check trees down the rows. Each test tree was bagged with about 50 apterous black cherry aphids taken from sweet cherry trees infected with Western X-little cherry. Every other check tree was bagged with the same number of aphids taken from presumably healthy cherry trees in a commercial orchard. The other check trees were not bagged.

In the tests conducted at Farmington, between May 1 and June 6, there was high mortality of both test and check trees. Of 600 trees in the plot 100 test trees and 65 check trees were dead. Apparently many of these trees died because of unfavorable growing conditions early in the season. The dead check trees included both trees that had and had not been exposed to aphid feeding.

In the tests conducted at Logan, from June 6 to June 19, nine of the 100 test trees wilted and died, but only one of the check trees. This tree had been exposed to the feeding of aphids from presumably healthy cherry trees. Other check trees apparently were not affected in any way by the virus-free aphids that were allowed to feed on them. This was also true of the 91 non-reacting test trees that had been exposed to the experimental aphids. These results strongly indicate that a virus transmitted by the aphids was responsible for the wilting and death of most, if not all, of the nine trees, and that toxins introduced by the aphids were not involved.

Nearly all of the dead check trees and some of the dead test trees observed at Farmington had not become established after planting. The tests at Logan were made after the trees had had sufficient time to become well established. Here it was possible to cull out all undesirable trees, and those used in the tests were in a vigorous condition at the time of exposure to the aphids and until the expression of wilt.

Although these results appear to indicate that the black cherry aphid is able to transmit Western X-wilt and decline disease to cherry trees on mahaleb rootstocks, more work is needed to confirm this conclusion. A report is being made at this time to make the information available

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[1]The authors are indebted to B. L. Richards and G. W. Cochran, Utah Agricultural Experiment Station, and B. N. Wadley and L. C. Cochran, U. S. Bureau of Plant Industry, Soils, and Agricultural Engineering, for helpful suggestions and aid in the diagnosis of wilt and decline in inoculum and experimental material. The work reported in this paper was financed with funds authorized under the Research and Marketing Act.

to investigators in other areas where this disease exists in cherry trees along with the Western X-virus complex.

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OCCURRENCE OF TOBACCO ETCH VIRUS ON GREENHOUSE TOMATOES IN OHIO

S. P. Doolittle [1] and L. J. Alexander [2]

In the fall of 1950 a number of plants in a large greenhouse at Toledo, Ohio, showed symptoms of combined infection by tobacco mosaic virus (Marmor tabaci Holmes) and cucumber mosaic virus (M. cucumeris Holmes). This virus complex frequently causes serious losses on fallcrop tomatoes in the greenhouse (1). Some plants showed also brown necrotic spotting of the leaves not characteristic of infection by this virus complex and of a type not previously observed by the writers. The same symptom was found later in other greenhouses at Toledo and in one at Columbus.

This necrotic spotting first appeared on leaves just below the growing point and usually only four or five leaves were affected. The spots were reddish-brown and of irregular outlines (Fig. 1, A). They were 1/8 to 3/8 inch in diameter and especially numerous near the margins of

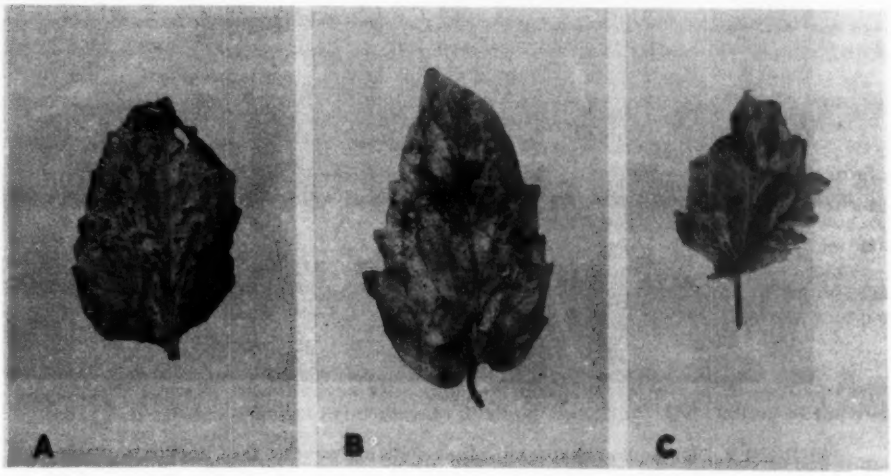

FIGURE 1. Leaflets of tomato plants infected by a combination of tobacco mosaic, cucumber mosaic, and tobacco etch viruses. (A) leaflet with typical necrotic spotting. (B) Leaflet showing necrotic spots and orange-yellow areas. (C) Leaflet of later growth showing mild green mottling and raised darker green areas but no necrosis.

the leaflets. Severely affected leaves showed so much marginal spotting that they had a scorched appearance (Fig. 2). Some of the spotted leaflets also showed blotches of an orange-yellow color with a faintly russeted appearance (Fig. 1, B). The spots were darker and more numerous than those caused by the leaf-withering strain of tobacco mosaic virus (Marmor tabaci var. siccans Doolittle and Beecher) (2) and were unlike those associated with the double-virus streak caused by combined infection by tobacco mosaic virus and potato virus X (Annulus dubius Holmes). Plants with symptoms of infection by both the cucumber and the tobacco mosaic virus showed severe necrotic spotting of the leaves. Fruits of these plants generally showed pronounced malformations like those that occur in plants infected by the cucumber mosaic virus. A similar necrosis was noted also on plants showing symptoms of only tobacco mosaic, but the spots were less numerous and much smaller.

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FIGURE 2. Greenhouse tomato plant showing severe leaf necrosis caused by combined infection by tomato mosaic, cucumber mosaic, and tobacco etch viruses. Photo by E. B. Wright, Jr., Wright Brothers Greenhouse Co., Toledo, Ohio.

The plants were not markedly stunted and after two to three weeks there was no further development of necrotic symptoms. As growth continued, there generally was a mild green mottling of leaves and occasional leaflets were abnormally broad and irregular in outline with rather large raised darker green areas (Fig. 1, C). The flower clusters produced on the portions of the stem bearing necrotic leaves set almost no fruit. However, blossoms produced on the later growth of plants with no evidence of infection by the cucumber mosaic virus set a fair number of fruit of normal size and appearance. In the greenhouse where it first was observed, leaf spotting was confined to plants in one section. There was evidence of spread down the rows in which diseased plants first occurred, but spread was not rapid.

Since it appeared that the necrotic spotting probably was the result of a combined virus infection, inoculations were made in greenhouses of the Plant Industry Station, Beltsville, Maryland, with juice extracts of diseased leaves on a number of host plants susceptible to some of the viruses commonly affecting tomatoes. The juice extracts were obtained from six tomato plants collected in one greenhouse at Toledo, Ohio, and from single plants from another greenhouse at Toledo and one at Columbus, Ohio. Six of these plants showed severe leaf necrosis. The other two had only a trace of such symptoms.

The inoculation tests showed that the six plants with severe leaf necrosis were infected by a combination of tobacco mosaic virus, cucumber mosaic virus, and tobacco etch virus (Marmor erodens Holmes). Only the tobacco mosaic and etch viruses were recovered from the two plants that showed only a slight spotting of the leaves. So far as the writers know this is the first occurrence of tobacco etch virus on greenhouse tomatoes in Ohio.

The tobacco mosaic virus isolated from all the specimens produced green mottling of the ordinary type on Turkish tobacco. All caused typical green mottle of tomato leaflets with little curling or deformity of the leaflets and no leaf necrosis.

The isolates of cucumber mosaic virus from tomato plants showing the same type of necrotic leaf spotting differed considerably in the severity of symptoms they produced on Turkish tobacco and tomato. Isolates from three of five specimens from a single greenhouse at Toledo caused a very mild mottling and no marked leaf deformity in tomato. On tobacco, the symptoms they produced were extremely mild and not typical of those produced by the ordinary strain of cucumber mosaic virus. Another tomato plant in the same greenhouse yielded an isolate that caused typical symptoms of cucumber mosaic on tobacco and extremely severe "shoe-string" malformation of tomato leaflets. The other isolates caused mild but typical symptoms on tobacco and tomato. The cucumber mosaic virus recovered from the plant from Columbus caused severe malformation of tobacco leaves but only mild distortion of leaves of tomato. On cucumber, the symptoms caused by the various isolates showed less variation and were similar to those produced by the typical cucumber mosaic virus.

There was little difference in the symptoms produced by tobacco etch virus recovered from the various tomato specimens. On tobacco, the etch necrosis caused by all the isolates was not very severe. On Datura stramonium the isolates all caused very severe malformation of the leaves. Symptoms in this respect were more pronounced than those caused by the etch virus recovered from peppers in eastern Maryland and Delaware. On tomato, the isolates all caused mild mottling with little curling or deformity of the leaflets.

The tobacco etch virus was readily recovered from leaves showing necrotic spotting but appeared to be in a low concentration in leaves taken farther up the stem where there were no necrotic symptoms. It is possible that the lack of necrotic symptoms on leaves produced some time after the necrosis first appears may be due to reduced activity of the etch virus in plants in which the tobacco or cucumber mosaic virus or both are present.

Some inoculations were made on young tomato plants in the greenhouse with various combinations of isolates of the three viruses. Tobacco etch virus plus cucumber mosaic virus caused yellowing of the older leaves similar to that seen on specimens from commercial houses. The yellowed leaves developed rather large, reddish, necrotic spots without sharply defined margins but such spotting was found on only a few leaves. The later growth of the plants showed only a mild mottling and little deformity of leaflets.

Tobacco etch virus combined with tobacco mosaic virus produced yellowing of older leaves of a few plants, and some of these leaves showed a few small, dark, necrotic spots. Plants infected by these two viruses showed pronounced mottling, dwarfing, and curling of the young leaves.

A combination of the three viruses in tomato produced yellowing and necrotic spotting of older leaves very similar to those occurring as a result of the combined infection by tobacco etch and cucumber mosaic viruses. The symptoms were not identical with those found on plants in commercial greenhouses but were like those produced on young tomatoes inoculated with juice from plants carrying the three viruses in natural combination.

These results appear to confirm observations made in the commercial houses where plants showing definite leaf and fruit symptoms characteristic of cucumber mosaic were most severely affected with necrotic spotting of the leaves. Where necrosis of the leaves occurred on plants showing symptoms of tobacco mosaic only, necrotic spotting seemed to be less severe.

It can hardly be assumed that the appearance of tobacco etch virus in Ohio greenhouses last fall represents the first occurrence of the virus, but it has not previously caused sufficient damage to be noticed by growers. We have no clue as to the source of the etch virus, but is it probable that it occurred on some cultivated crops grown in market gardens in the vicinity of the greenhouses. As far as we know, the etch virus was not prevalent in the Cleveland area, where there is a large acreage of vegetables under glass, but it occurred rather commonly at Toledo and was present in at least one greenhouse at Columbus. As yet there have been no reports of such infection on the spring crop.

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OVERWINTERING OF PHYTOPHTHORA PHASEOLI ON INFECTED LIMA BEAN PODS IN THE SOIL

R. S. Cox and R. A. Hyre

Experiments were set up in 1950 to determine if, and in what manner, the lima bean downy mildew fungus overwinters in Delaware. This note is to report that positive results have been obtained; details will be presented later. This is believed to be the first concrete evidence as to how the fungus overwinters.

Downy mildew-diseased lima bean pods were placed either (1) on the surface or (2) buried two inches in steamed and field soil in 10-inch pots which were placed outdoors for the winter. At intervals pots of the different series were brought in to the greenhouse to controlled (cool, moist) atmospheric conditions and planted with lima bean seed.

In addition to the pot series overwintering tests were made using field plots. Diseased debris was plowed under in the fall, in the spring, and disced in at seeding time on different plots.

Downy mildew was found on July 1, 1951, on plants grown in pots with buried diseased pods that had been overwintered outdoors and brought in to the greenhouse and seeded on June 5. Infected plants exhibiting the sporulating fungus were found, also, on July 12 in the field plot where diseased refuse was plowed under the preceding fall. Infection has not been found in controls of either the greenhouse or field plantings to date (July 16). No downy mildew was found during a survey on July 16 of the commercial lima bean areas of the State. Hot dry weather conditions had prevailed throughout these areas during July.

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WATERMELON DISEASES IN THE GAINESVILLE AREA OF FLORIDA FOR THE 1951 SEASON

George F. Weber and John H. Owen

Fusarium wilt (Fusarium oxysporum f. niveum) is the most prevalent and destructive disease in the Gainesville area, this season. A dry season becoming more acute as the crop matures, combined with high temperatures, contributed to the large losses due to wilt, and infection ranged from 1 to 5 percent on new land to 10 to 50 percent of the plants killed on old cultivated land.

The following rainfall was recorded in this area for the first 6 months of 1951.

Month	1951	48-Year Average Rainfall
January	1.07 inches	3.16 inches
February	1.05	2.92
March	5.00	3.35
April	2.16	2.40
May	2.31	3.15
June 15	.45	3.42
Total	12.04 inches	18.40 inches

The rainfall thus far in 1951 is 30 percent less than the 48-year average. Precipitation during the month of March is adequate; however, the plants were just coming up at this time. The June pre-harvest period was dry and hot, thus increasing the amount of wilt damage.

Anthracnose (Colletotrichum lagenarium). Although dry weather prevailed during most of the growing season, the anthracnose fungus produced as much as 10 percent infection on the stems in one 50-acre field and less than 1 percent infection in three other fields. No leaf or fruit symptoms were observed.

Gummy-stem blight (Mycosphaerella cucumis) was observed in four fields, occurring in the crown of the plant, and in all instances was producing less than 1 percent infection.

Leaf spot (Macrosporium cucumerinum) [Alternaria cucumerina] caused severe damage to foliage in most of the fields, with infection ranging from 1 to 75 percent. This disease appeared in scattered local areas in the fields, killing the foliage and vines. It ranks second to wilt in the amount of damage occurring this season.

Downy-mildew (Pseudoperonospora cubensis) was not observed in any of the fields.

Acres in field	Disease (Percentage)				
	Fusarium	Alternaria	Anthracnose	Gummy-stem blight	Downy mildew
50	0	5	10	0	0
50	10	1	0	0	0
30	5	5	0	0	0
12	2	2	1	1	0
25	50	2	0	1	0
20	20	0	0	0	0
40	1	60	1	1	0
30	20	10	1	1	0
21	30	5	0	1	0
10	0	0	0	0	0
80	2	75	0	0	0

COLLEGE OF AGRICULTURE, UNIVERSITY OF FLORIDA, GAINESVILLE, FLORIDA

INEFFECTIVENESS OF METHYL BROMIDE FOR KILLING THE CAUSAL ORGANISMS OF RING ROT AND SEED PIECE DECAY OF POTATOES

W. F. Mai, Bert Lear, and C. W. Boothroyd

Corynebacterium sepedonicum (Spieckermann & Kotthoff) Skapt. & Burkh., the causal organism of ring rot of potatoes, and Fusarium sambucinum Fckl., one of the species of the genus Fusarium capable of causing potato seed piece decay, can be spread from farm to farm on burlap bags. Treatment of bags by immersion in hot water or dipping in chemical solutions results in rotting unless the bags are rapidly and thoroughly dried after treating. A treatment lethal to both of the above organisms, and especially to the ring rot bacteria, but non-injurious to bags is urgently needed because of the scarcity and high price of burlap.

Methyl bromide has been reported toxic to the bacterium causing ring rot at the rate of 19 pounds per 1000 cubic feet for 16-20 hours [1]. A volatile chemical such as methyl bromide would be particularly suitable for treating burlap bags because it penetrates rapidly and disappears quickly from closed chambers after the exposure period. Because of the possibility of its use as a practical method for treating burlap bags, extensive tests were made of the toxicity of methyl bromide to C. sepedonicum and F. sambucinum.

Table 1. Toxicity of methyl bromide to Corynebacterium sepedonicum.

Treatment		Number tomato plants inoculated	Number tomato plants showing ring rot symptoms
lbs./1000 cu. ft.	Duration hrs.		
46	2	60	60
23	16-20	60	47
46	16-20	50	50
60	16-20	20	20
No treatment		60	59

Table 2. Toxicity of methyl bromide to Fusarium sambucinum.

Treatment		Number toothpicks contaminated	Number toothpicks on which organism survived
lbs./1000 cu. ft.	Duration hrs.		
46	2	19	17
23	16-20	20	18
60	16-20	20	15
No treatment		20	20

The toxicity to the ring rot organism was tested according to a method developed by Gonzenbach [1]. Round toothpicks were contaminated by contact with infected tuber tissue. After drying for 24 hours, the contaminated toothpicks were exposed to the desired methyl bromide treatment in a fumigation chamber constructed from a large pressure cooker. In one test, involving the highest dosage and longest exposure period, the union between the lid and pan of the pressure cooker was sealed with heavy cellophane tape to make certain that gas was not escaping. After aeration for 24 hours, the contaminated end of each toothpick was forced into the stem of a small Bonny Best tomato plant. The tomato plants were observed over a period of three months for the appearance of ring rot symptoms.

Increasing the dosage or lengthening the exposure period resulted in slower development of the ring rot symptoms in the tomato plants. Symptoms developed at a more rapid rate when the tomato plants were small at the time of inoculation.

[1] Gonzenbach, Corlos Tirzo. A biologic method for testing chemicals for the control of the potato ring rot pathogen, Corynebacterium sepedonicum (Spieck. and Kotth.) Skapt. and Burkh. Thesis, Cornell University, Ithaca, N. Y. 1949.

The toxicity of methyl bromide to F. sambucinum was tested by an adaptation of Gonzenbach's method. Toothpicks were contaminated by contact with sporulating cultures of this organism growing on potato dextrose agar. The contaminated toothpicks were allowed to dry, exposed to methyl bromide in the fumigation chamber, aerated, and then placed on potato dextrose agar. When the fungus was not killed by the treatment, colonies appeared on the agar in from two to four days.

The results of all treatments are recorded in Tables 1 and 2. Dosages of methyl bromide up to 60 pounds per 1000 cubic feet for 16-20 hours exposure at temperatures ranging from 72 to 80 degrees Fahrenheit were ineffective in killing either of these organisms. Longer exposure periods or higher dosages were not considered practical.

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THE DISTRIBUTION OF TOBACCO BLACK SHANK IN NORTH CAROLINA IN 1949 AND 1950

C. J. Nusbaum

In recent years the incidence of black shank, caused by Phytophthora parasitica var. nicotianae, has increased in North Carolina at an alarming rate[1]. Since 1946, the number of farms invaded by the black shank pathogen has nearly doubled every year. The disease has been found in 54 of the 61 counties producing flue-cured tobacco. About one-fifth of the flue-cured tobacco farms in the State now have fields infested with the causal organism. In areas centering on Forsyth County in the Old Belt and Pitt County in the east, where black shank appeared in the 1930's, most growers now find it necessary to plant resistant varieties.

Tobacco production, marketing, and utilization, as well as research and extension programs, are greatly influenced by black shank. In fact, practically everyone connected with the tobacco industry in North Carolina is interested in the disease. Black shank causes heavy losses every year, especially on farms where the disease occurs for the first time, because susceptible varieties invariably are grown on such farms. It is also a threat to growers who have not yet encountered it. The disease is bringing about rapid changes in the variety situation. This naturally is of concern to growers interested in the field performance of new sorts and to those interested in their quality and suitability for the manufacture of tobacco products.

Yearly surveys, based upon reports from county agents and extension specialists[2], give a reasonably accurate estimate of black shank distribution. These data also indicate the pattern of spread from year to year and furnish a basis for anticipating future spread in certain areas. Such information also is useful to seedsmen in estimating seed requirements and to extension personnel in planning educational programs.

Table 1. The distribution of Phytophthora parasitica var. nicotianae in certain flue-cured tobacco producing areas in North Carolina in 1949 and 1950.

Belt	Area	Number of farms infected		
		1949	1950	Percent increase
Old	Forsyth County*	1,603	1,863	16
	Counties adjacent to Forsyth	5,962	9,129	53
	Others	1,523	3,320	118
Middle	Entire	744	2,433	227
Eastern	Pitt County**	2,400	2,800	17
	Counties adjacent to Pitt	2,257	4,135	83
	Others	832	3,355	303
Border	Entire	0	15	
Total		15,321	27,035	76

* Black shank was first found in North Carolina in Forsyth County in 1931.
** A new center of black shank infection was found in Pitt County in 1937.

The results of surveys conducted in 1949 and 1950 are given in Table 1. An outline map of North Carolina, identifying the various belts and areas referred to, is shown in Figure 1.

Black shank has occurred every season since 1931 in Forsyth County, the original focus of infection in North Carolina. From this point, the infestation has spread and also has become intensified in the central part of the area. In Forsyth County and some adjacent areas, the infestation has more or less reached the saturation point. Most of the soils used for tobacco production there are infested with the pathogen and resistant varieties are grown extensively. The farm

[1]Moore, E. L., et al. Black shank and Granville wilt in North Carolina in 1948. Plant Dis. Reptr. 33: 183-186. 1949.

[2]H. R. Garriss, extension plant pathologist, R. R. Bennett, extension tobacco specialist, and S. N. Hawks, assistant extension tobacco specialist, N. C. State College, have given valuable aid in obtaining survey data.

OLD BELT
MIDDLE BELT
EASTERN BELT
BORDER BELT
★ - Forsyth County. First report of black shank in 1931.
⊙ - Pitt County. First occurrence of black shank in Eastern Belt in 1937.

FIGURE 1. Outline map of North Carolina showing the flue-cured tobacco belts and the location of the first outbreaks of black shank in Forsyth and Pitt Counties.

infestation in Forsyth County increased only 16 percent from 1949 to 1950. The increase in the number of infested farms in adjacent counties from 1949 to 1950 was 53 percent, indicating the degree of intensification that is taking place. Here the infestation is rapidly approaching saturation. In other counties of the Old Belt the black shank fungus is well established and is building up rapidly. This is shown by the increase of 118 percent in the number of infested farms in 1950 over 1949.

From the Pitt County area infection center, established in 1937, the infestation has spread in a manner similar to that observed in the area centered on Forsyth County. In Pitt County itself, the infestation is nearing saturation and the rate of intensification is apparently declining. Here the increase in the number of infested farms in 1950 was 17 percent. However, considerable intensification is taking place in adjacent counties (83 percent increase) and even to a greater extent in the remainder of the Eastern Belt counties (303 percent increase).

Before 1947, the Middle Belt was practically free of black shank. Spread of the pathogen into this area probably took place from both Old and Eastern Belts. Thus it may be represented as a middle zone in which the advancing margins of the two large centers of infestation met and overlapped. In this area intensification is now taking place at an accelerated rate as shown by the nearly fourfold increase in the number of infested farms in 1950. In Wake County no black shank was found prior to 1948. In that year the disease was found on 11 farms. In 1949 the number of infested farms rose to 126 and in 1950 to more than 800.

In the Border Belt, black shank appeared for the first time at several scattered locations in 1950. The infestation probably is the result of southwestward advance of the black shank fungus from the Eastern Belt.

The growing prevalence of black shank has complicated the tobacco disease situation in certain areas, as fields become infested with the causal organisms of two or more major diseases. Bacterial wilt, caused by Pseudomonas solanacearum, is prevalent in certain areas of the Middle, Eastern, and Border Belts. Fusarium wilt, caused by Fusarium oxysporum var. nicotianae, is an important disease in the Border Belt and in the southern part of the Eastern Belt. In 1949, 844 farms had fields infested with the causal organisms of both black shank and bacterial wilt. The number of such farms increased to 1546 in 1950. Thus far, an association of black shank with Fusarium wilt has been found on only a few farms. In 1950, two cases were observed where all three of these diseases occurred on the same farm.

The history of black shank in the flue-cured tobacco belts of North Carolina gives a rather clear picture of the spread of the causal organsim. After the pathogen becomes established in a new area, the inoculum potential at first is low and spread is slow. Within a few years new infestations appear near the original focus. The inoculum potential then increases rapidly and

the rate of spread begins to accelerate. The continued use of susceptible varieties in fields not yet invaded by the black shank fungus favors rapid spread.

If the present pattern of spread and intensification continues, it is anticipated that the infestation soon will reach saturation in Forsyth and Pitt Counties and also in counties adjacent to them. Infestation in other Old and Eastern Belt counties and also in those of the Middle Belt is probably approaching the peak of an accelerated rate of spread. In the Border Belt it may take three or four years for the infestation to build up sufficiently to begin a period of rapid buildup. In fact, the present degree of infestation in the Border Belt is similar to that observed in certain Middle Belt areas three years ago.

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HOP CANKERS

C. E. Yarwood

In 1935, 1942, 1946, 1947, 1948, and 1949 cases of hop canker in Sonoma, Mendocino, Lake, and Sacramento Counties were called to the writer's attention.

The most important type (Fig. 1) usually extended about three inches above and three inches below the ground line of trained vines, and was never observed to extend to the main root. Rarely, cankers of which the lower margin was above the ground line were seen. The primary symptoms were a browning of the cortex in limited areas. Later the infection girdled the stem, possibly by fusion of smaller infected areas. Liquid exudation from the stem usually followed and sometimes this became foamy, apparently owing to the action of microorganisms within the exudate. Sometimes the exudate soaked into the string on which the vine was trained, and caused a growth of mold. Of the organisms on the string only Penicillium was identified. Later the infected tissue collapsed to less than the diameter of the normal vine (Fig. 1B). Rarely (only two cases observed) the cortex and pith were apparently completely destroyed, leaving only a shredded vascular system in the canker area, yet the vines above did not die, and complete killing of the vine above a canker has not been observed. Usually the cankers healed over with a considerable increase in the diameter of the vine but with splitting of the cortex (Fig. 1, C, D). Top symptoms were a temporary wilting of the vine, yellowing of the lower leaves, and retardation of growth. The first seasonal record of the disease was June 16, but new records of the disease were as late as August 15.

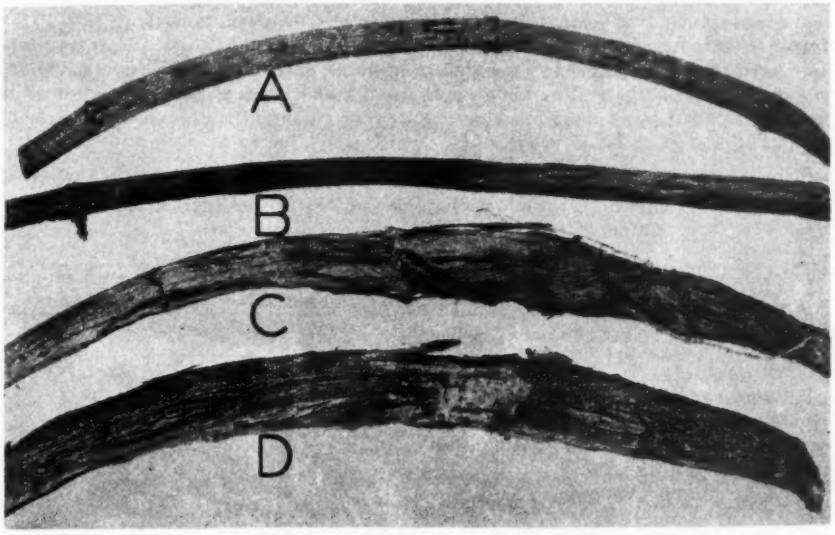

FIGURE 1. Hop canker. A, base of healthy vine. B, medium stage of canker showing blackening of surface, and depression of central canker area. C, advanced stage of recovered canker showing thickening of stem and splitting of cortex. D, as C but also showing Fusarium sporulation in central area of canker. Photographed August 23, 1946, by Victor Duran.

In the early stages of the disease no fungus fruiting on the surface was observed, but a Phytophthora or Pythium (not identified) was consistently isolated. In later stages of the disease (Fig. 1, D) a Fusarium, identified as F. roseum by Dr. H. N. Hansen, was commonly sporulating on the surface of the cankers, where the pink spore masses could be seen with the naked eye. Dr. Hansen also isolated from old cankers a Phytophthora or Pythium which was apparently the same as that isolated from young cankers. Dr. Hansen considers the Fusarium to be a saprophyte and inoculations with this organism were not attempted. Field inoculations with young pure cultures of the Phytophthora or Pythium under apparently favorable conditions for infection were not successful.

Hop canker as seen in California has been a minor disease. Though no yield records were taken it is believed that infected vines yielded at least 75 percent of the yield of normal vines.

In most yards and most seasons the disease has not been found. The maximum occurrence has been 41 vines with canker out of 100 counted in a yard at Hopland on August 6, 1947. The average of all other natural occurrences seen by the writer (excluding all cases where the disease was not found) was 7 percent of the vines with canker. Usually only one vine per hill was affected, but occasionally all vines in a hill (up to four vines per hill) were attacked. Though no adequate surveys were made, the writer believes that for the period 1935 to 1950, the incidence of the disease in California has been less than 1 percent of the vines infected, and less than a 1/4 of 1 percent crop loss.

All severe cases of the disease which have come to the writer's attention have been associated with a combination of stripping, suckering, hilling, and irrigation, which are each normal procedures practiced in the sequence indicated one to three times per season. Stripping is the removal of the lower laterals from the trained vines. Suckering is the removal of all other than trained shoots from the crown of the hill. Suckering and stripping are usually performed in one operation. Hilling is commonly practiced as an aid to basin irrigation, and also for weed control and for protection of the crowns. Irrigation is either by the basin method, by rows, or by overhead sprinklers, the last of which are increasing in use. When overhead irrigation is practiced hilling is less common.

In one yard in Sacramento County in 1947 the disease was abundant where stripping, suckering, hilling, and row irrigation had been completed a week earlier but was absent in otherwise similar portions of the yard ahead of these operations or where only one or two had been completed. It seemed likely that canker infection had entered through wounds made in stripping and suckering, that inoculum came from a reservoir in the soil which was piled against the wounds in the hilling operation, and that subsequent irrigation had maintained a moist condition of soil and stem tissue long enough for the pathogen to oecome established.

In trials to determine the role of stripping and suckering, hilling, and irrigation in canker formation, artificial wounding was resorted to in some units. Wounds were made one per vine just above the normal soil level by scratching the trained vines with a knife over an area about 1 cm. long and about 1 mm. deep, and leaving frayed tissue at each end of the wound. Treatments of May 26 and June 2 and results of June 16, 1950, were as shown in Table 1.

Table 1. Results of tests to determine effect of various cultural treatments, with and without wounding, on the incidence of hop canker.

Treatment	Number of vines treated	Percent of vines with canker
None	300	0.7
Hilled only	180	1.7
Watered only	40	0
Stripped and suckered only	140	0
Injured only (scraped with knife)	40	0
Hilled and watered	40	12.5
Injured and watered	40	2.5
Stripped and suckered, hilled, and watered	80	9.
Injured, hilled, and watered	40	48
Injured, stripped and suckered, hilled, and watered	80	29

The cankers produced in these ways seemed similar to those which occurred naturally. Of the four factors under study, only the hilling, when applied alone, caused any infection. In combination with watering, hilling was more effective in producing infection than was artificial injury. All combinations of two or more treatments produced some disease, but several important combinations of treatments, necessary for a proper evaluation of the role of each treatment, were omitted. From the available evidence, however, the writer believes that the decreasing order of the importance of the tested treatments in bringing about hop canker were: hilling, watering, injury, and stripping and suckering. It seems likely that the disease could be almost avoided by elimination of hilling. Elimination of irrigation is not practical but it would seem likely that if hilling is practiced, stripping and suckering should precede hilling by a period long enough for the wounds to heal.

On the basis of the general symptoms and the presence of Fusarium spores in the cankers, the disease was originally diagnosed as hop canker caused by Gibberella sp. as described in England (3, 4, 5). The regular presence of a Phytophthora or Pythium in the cankers makes this diagnosis doubtful. On the basis of the location of the cankers, the foaming of the exudate, and the nature and weak pathogenicity of the fungi associated with the cankers, the writer believes the hop canker described here resembles quite closely the foam disease of citrus (1).

The second type of hop canker, even less important than the first, presents the same symptom picture as that described in Oregon by Hoerner (2). It has been seen by the writer only twice, once in Sonoma County in 1942 and once in Sacramento County in 1947. It has been seen only in hot weather and at the points where trailing vines from apparently healthy hills rested on lumps of soil.

The third type of hop canker, seen only once in British Columbia in 1929, occurred at about breast height where the vines were tied together with string. One vine had a canker about three inches long and contained sclerotia of Sclerotinia sclerotiorum in the pith.

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ROOT KNOT OF PEANUT. I. DISTRIBUTION

J. H. Machmer

Peanuts are resistant to most species of the root-knot nematodes and have been used extensively and effectively in rotations as a root-knot resistant crop. There are, however, two species of root-knot nematodes in the eastern United States which will infect peanuts and cause serious crop losses in the regions where they occur.

As reviewed by Tyler (6), peanuts are reported by most plant pathologists as "highly resistant", and "resistant under a variety of conditions", to root knot, but there are rare observations of severe infections. In 1889, Neal (5) of Archer, Florida, wrote: "The roots of peanuts became masses of knotty roots, the worst case of the disease I ever saw." Root knot has also been identified on peanuts many times by the Division of Nematology, Bureau of Plant Industry, Soils, and Agricultural Engineering, but not until July, 1947, was it positively identified from Georgia.

In the peak production years, 1946-48, an area in south Georgia, southeast Alabama, and north Florida comprised about half of the Nation's commercial peanut acreage. In Georgia alone, at that time, 1 1/2 million acres were planted or interplanted to peanuts. Since the peak production years peanuts have been Georgia's numbers 2 and 3 field crop, approximating 70 million dollars income annually.

Of the two root-knot nematode species which infect peanuts, that reported by Christie and Albin (2) from Pitt County, North Carolina, has been assigned the name Meloidogyne hapla, Chitwood (1). Root knot of peanut has subsequently been reported in that State by Cooper (3) and is said to occur on peanuts in Virginia. M. hapla appears to be the prevailing root-knot nematode of the northeastern States and is widely distributed north and west beyond the Albemarle-Chesapeake peanut region.

The other root-knot nematode species which infects peanuts was identified as M. arenaria (Neal) Chitwood (1), from material collected in southwest Georgia by the writer. As reported by Wilson (7) and by Machmer (4) this root-knot nematode of peanut occurs over a considerable area in the lower Chattahoochee River basin where peanuts are the most important field crop.

No thorough survey has been made to ascertain the exact extent, geographical limits, or degree of root-knot infection of peanuts in the lower Chattahoochee River basin. The statements made herein are based on field observations and on limited surveys of failing peanut fields on several hundred farms in some 30 Georgia counties during the 1947 to 1950 pre-harvest and harvest seasons, and are supplemented by information obtained from various specialists and growers who have cooperated on the problem.

Root knot on peanuts in the lower Chattahoochee River basin occurs over a somewhat triangular area having sides of approximately 125 miles, with Webster County, Georgia, at its northern point. The triangle extends southeastward into Grady County, Georgia, then westward into Coffee County, Alabama. It occurs in ten related counties in Georgia and is known in at least four counties in Alabama. Over some of this area, as for example, Seminole, Decatur, Early, Clay, and possibly Miller and Grady Counties, in Georgia, the infestation is general with probably half the peanut acreage affected. About 30 percent of Georgia's 1,100,000 acres at present in peanuts lies within counties where root knot of peanut is known to occur. Approximately 300,000 acres of peanuts are grown in Alabama and most of this acreage lies in or near counties where root knot of peanuts has been reported. On the other hand, in Baker, Calhoun, Webster, and Stewart Counties of Georgia, only a few infested fields have been found, but in such areas further field observations are needed before any assurance can be given that the disease is not widespread. Only a few observations have been made in Jackson County, center of the western Florida peanut area, but no root knot was found. Likewise, root knot of peanut has not been reported in north-central Florida except that found by Neal over 60 years ago. Outside the lower Chattahoochee River basin it was collected from only one field in each of Dodge and Jefferson Counties, Georgia, in September, 1950.

It is possible that the root-knot nematode of peanut in the lower Chattahoochee River basin is indigenous, that it may not be restricted to the lower part of the basin, and that although ordinarily latent, it may occasionally become epidemic when conditions favor this behavior.

In 1949 and again in 1950 no root knot was found during many examinations of peanut fields showing suspicious symptoms, in a tier of counties immediately east of the Flint River, Georgia. Except for parts of Grady and Dougherty Counties, the Flint River appears to be the eastern boundary of the distribution of M. arenaria, the Chattahoochee peanut root-knot nematode. This may have a direct bearing on the use of peanut rotations for root-knot control in Georgia's tobacco belt which extends eastward from the Flint River. The root-knot nematode species found

on a few inconspicuously galled root-knot infected peanut plants collected in Dodge and Jefferson counties, locations far eastward and unrelated to the lower Chattahoochee River basin, have not been adequately identified. Further and more extensive field surveys may reveal the presence of one or several root-knot diseases of peanut in Georgia's peanut-tobacco belt.

Heavily galled peanut seedlings can be found in the lower Chattahoochee River basin in May. Top peanut vegetation in root-knot infested fields however, usually does not show conspicuous symptoms until the season approaches harvest. At this time plants are becoming discolored and are stunted so that they fail to cover the soil between rows. The slowly dying and browning plants present a mottled effect among the greener plants and weeds. When the roots of such plants show conspicuous root-knot galls the neighboring plants in apparent vigor are usually well infected also. Southern blight, on the other hand, appears as a rapid dying and blackening of individual plants or of long sections of a row.

Galling occurs on all underground parts of peanut plants including the pods which appear warty. Pod stems are often heavily galled and are easily severed. Early infection of the "peg" (ovary) is detrimental to the seed embryo. Heavily galled plants frequently have a great many necrotic "pegs" but less than a dozen mature peanuts.

Peanuts are not tolerant to Meloidogyne arenaria and are severely injured by it. A field where the disease is general and severe is often a total loss. Some fields are known on which harvest operations were abandoned because of root knot alone. The extent to which yields have declined is indicated by the comments of different growers. "Cannot make a 10 percent crop"; "Not worth harvesting"; "Cannot make an acre crop in five"; "Not a peck to a stack"; "There are no nuts or they are warted"; "Not worth hogging off"; "Didn't take up the crop last year"; "Been having trouble for five years"; etc. On a farm where the disease has been continually severe and has been closely observed since 1947, the 1950 crop from a 16-acre allotment brought only $356.16. Many fields of extremely sub-average yield (under 750 pounds per acre) could produce 1500 pounds (or $200) per acre, if root knot were controlled.

Peanuts, especially the Spanish types, in the lower Chattahoochee River basin, are frequently and severely affected by southern blight (caused by Sclerotium rolfsii Sacc., locally called white mold). Injury caused by southern blight had so masked symptoms of root knot that the latter disease most probably escaped wide detection. It was not until growers, between 1947 and 1949, changed 90 percent of the acreage from Spanish to runner types, which seemingly survive southern blight better, that the seriousness of root knot became apparent. The disease was further favored by the increased wartime peanut acreage which required one-half to two-thirds of the suitable peanut land to be planted successively to peanuts.

Coffee weeds, Cassia tora L. and C. occidentalis L., occur in the region as commonly persistent weeds and are repeatedly found severely galled in root-knot infested peanut fields, although they, like peanuts, have often been reported in the literature as highly resistant to root knot. Whenever Cassia species were found infected in southwest Georgia, the nearby peanut roots were always conspicuously galled.

Although neither corn nor cotton is resistant to many kinds of root knot, many varieties of both appear suitable rotation crops for control of this root-knot nematode of peanut. Hairy indigo (Indigofera hirsuta L.), widely used as a root-knot resistant rotation crop, proved very susceptible to the root-knot nematode of peanut on a southwest Georgia experimental planting. Many summer and winter peas, clovers, lupines and other legumes have been tested and all appear only to increase or sustain the infestation in peanut fields. Crotalaria species and beggar weed (Desmodium sp.), on the other hand, being immune, serve to reduce infestation, Other suggestions of rotation crops for use in root-knot control will be reported later. In the meantime the use of a weed-free, crotalaria-oats rotation for two years still remains the most effective control practice under south Georgia conditions.

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AN EXCEPTIONALLY EARLY OUTBREAK OF CORN RUST IN CANADA [1]

W. E. McKeen [2]

Urediniospores of Puccinia sorghi, the causal agent of corn rust, were observed as early as June 11, 1951, on corn leaves in southern Ontario. In 1950 and 1949 they were not detected until July 31 and August 25 respectively. These sori appeared only 20 days after emergence of the corn seedlings and thus infection was initiated while the seedlings were in the second or third leaf stage. The earliness of the appearance of this disease is of considerable importance because this fungus may repeat many more cycles than in previous years, with each cycle greatly increasing the potential inoculum and possibly its severity. If suitable environmental conditions prevail throughout this season an epiphytotic may develop which could be more disastrous than in 1950. Previous to this the pathogen subsisted without much indication that it would seriously affect corn.

Last season 100 percent of the plants in commercial fields were infected but the severity of rust infection varied a great deal. This variation was much more noticeable in the inbreds; a few appeared to be immune while others were destroyed by mid-August. Probably in future breeding work it might be advisable to select only resistant inbreds for hybrid production. It is practically impossible to say whether or not a new race or races, or the subnormal summer temperatures and high average rainfall, or the overwintering, or a combination of these were responsible for the epiphytotic. Because of the potential threat of this disease throughout the whole corn belt of North America and the dearth of fundamental information concerning control measures and factors influencing corn rust epiphytotics, some urediniospore germination studies were conducted at Harrow, Ontario.

Germination proved to be excellent at temperatures of 13° to 27° C. but at 31° C. germination dropped to less than 1 percent. This indicates that continuous hot weather would retard the development of this organism.

Weekly or fortnightly germination tests in Van Tieghem cells, of urediniospores obtained from infected leaves which were taken from corn shocks and corn in an open barn, showed that viability gradually decreased throughout the winter until May 7. At this latter date only a trace of spores produced germ tubes, and on May 23 and 25, the time of corn emergence, spores from a sorus from each of ten different leaves on each date failed to show any signs of germination. Whether infection was initiated by an overwintered urediniospore or by an aeciospore which came from the alternate host, Oxalis spp., is debatable. The chance of survival of urediniospores in the exposed fields is much less than in protected areas and thus the source of this inoculum remains uncertain since no aecia have been located this season.

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CONTROL OF THE LEAF BLIGHT AND RUST DISEASES OF SWEET CORN

G. R. Townsend

Leaf blight (Helminthosporium turcicum) has been very troublesome for growers of sweet corn in the Lake Okeechobee section of Florida for several years. Rust (Puccinia sorghi) was first noted to be severe in a few fields of hybrid sweet corn in the spring of 1950. The experiment reported here was designed to test formulations and schedules for the application of Dithane (zineb) dusts to control the leaf blight disease. The experiment was conducted at Belle Glade, Florida, in the spring of 1951. Not only did it give information on the control of the Helminthosporium blight disease, but data also were obtained on the control of the corn rust.

Hybrid sweet corn of the F. M. Cross variety was planted for the trial on February 21, 1951. The field was laid out in such a manner that 4-row plots could be dusted with a high-clearance duster. There were four 4-row plots in a randomized block arrangement for each treatment. Four barrier rows were planted between each of the plots that were to receive the treatments in order to minimize the effect of drifting dust. In addition a further precaution was taken in that the data were taken from only the two center rows of the dusted plots. The fungicidal formulations were dusted on the corn at the rate of approximately 50 pounds per acre in either seven or eleven applications over a period of 55 days between March 6 and April 30, 1951. DDT was used in all of the dust formulations and alone on the checks when necessary. The schedule of fungicide treatments tested is shown with the tabulation of the data.

Table 1. Summary of data on the control of sweet corn rust and blight in field trials at Belle Glade, Florida, 1951.

Treatment	Disease ratings[a]		Boxes per acre[b]			Percent	Weight per dozen ears[c]
	Blight	Rust	No. 1 ears	No. 2 ears	Total	No. 1 ears	
1. No fungicide	3.50	4.00	105	75	180	58.4	6.96 lbs.
2. 10% Z-78 dust 11 applications in 55 days	1.00	1.00	163	62	225	72.5	7.80
3. 10% Z-78 dust 7 applications in 55 days	3.00	2.25	150	53	203	73.9	7.80
4. 10% Z-78 dust 7 applications, none in last 25 days	1.75	1.75	162	49	211	76.8	8.16
5. 10% Z-78 dust 7 applications, none in first 30 days	1.75	2.00	160	47	207	77.2	8.04
6. 5% Z-78 dust 11 applications in 55 days.	1.50	1.75	165	55	220	75.0	8.04

[a] Disease ratings: 1- light; 2- moderate; 3- heavy; 4- severe.
[b] Boxes per acre: Factor, 0.52 x number of ears picked from four plots; gives yields in boxes of 5 dozen, with rows spaced at 36 inches.
[c] Weight per dozen ears: Total weight from four plots divided by total number of ears and multiplied by 12.

Rust was first observed on March 23 and soon became so severe on the check plots that it was obvious that differences would be obtained. The progress of the rust disease slackened as the weather became warmer in late April, but this disease had already done considerable damage as the leaves on the worst affected plots were beginning to dry out at this time. The Helminthosporium disease was slow in starting because the early season was cool and dry, but

frequent showers in April improved conditions for infection and the disease was showing by the end of the month, and developed rapidly during the first week of May when the corn was becoming mature. Individual scores for each disease were made on all of the plots on May 8. The average disease reading for each treatment and each disease is shown in Table. It is felt that the rust disease accounted for somewhat greater damage to the corn than the blight as it appeared earlier.

The results of this experiment are summarized in the Table. It is shown in the data that both diseases were well controlled by the application of Dithane dusts where the schedule was suitable. The disease ratings were lowest where the 10% dust was used regularly through a schedule of eleven applications. However, the 5% dust on the same schedule was nearly as good, and certainly was more economical since the difference in yield was only five crates per acre. The control of rust and blight by these treatments increased the yield of number one grade corn by about 60 boxes per acre, and the total yield was increased by about 40 boxes per acre.

The omission of part of the applications over the whole schedule resulted in poorer control and a lower yield. On the other hand when the fungicide was applied in only seven of the early applications, control was nearly as good as with the full schedule. Control was not quite as good when four early applications were omitted, and the seven later applications were made.

The data on the percentage of ears falling in the number one grade (well filled and over 6.5 inches long) show that the control of rust and leaf blight increased the number of ears falling into these grades. The ears from the controlled plots weighed more than ears from the check plots. These data are indicative of an up-grading for quality due to the control of the two diseases.

The economic gain to be expected from the control of rust and blight in corn would result from obtaining a higher price for the higher quality corn, and from a greater total yield. By applying prevailing prices to the data from this experiment it can be shown that the gain for the treatments varied from $68.50 to $119.00 per acre, less the cost of the dusts. The most economical schedule therefore would be the use of the 5% dust on the full schedule, or the 10% dust for the applications up to the time the corn starts to tassel.

BELLE GLADE, FLORIDA

RACE 55 OR 101 OF CROWN RUST OF OATS WHICH ATTACKS VICTORIA AND BOND DERIVATIVES

H. R. Rosen and H. C. Murphy

Recently the writers (Phytopath. 41: 31, 1951) called attention to a race of crown rust (Puccinia coronata) apparently new to the United States which appeared "to be somewhat similar to race 55 previously found in Argentina". Because of the serious threat that this race seemingly offers to both Victoria and Bond derivatives as well as to Red Rustproof strains, it appears desirable to present additional information on the identity of this race and on some other important aspects.

This new race may be designated as race 55 or 101 depending upon the reliance placed on the reactions of the differential variety, Ruakura. Vallega (Informaciones de interes para los trabajos de seleccion de avenas resistentes a Puccinia coronata avenae. Inst. de Fitotecnia, Buenos Aires Hoja informative No. 2, 4 pp. 1945) who first found and described race 55 (in Argentina), considered Ruakura to be resistant to this race, whereas the present writers have found it to be susceptible to collections which otherwise produce reactions on all other differential varieties very similar to those described by Vallega for race 55. Nevertheless, since race 1 is distinguished from race 6 on the basis of difference in Ruakura reactions and race 45 is likewise distinguished from race 57 on the same basis, to be consistent races 55 and 101 would have to be recognized as distinct races. Because of the variability in reaction of Ruakura to any one race, this variety probably will be dropped from the list of differentials for future race identification work.

The mean type reaction of race 101 on all the differential varieties utilized up to the present for the identification of races of crown rust, on seedlings kept at approximately 70° F. in a greenhouse, is as follows:

Ruakura 3 (susceptible)
Green Russian 3 (susceptible)
Hawkeye 4 (completely susceptible)
Anthony 4 (completely susceptible)
Sunrise 4 (completely susceptible)
Victoria 3 (susceptible)
Green Mountain 4 (completely susceptible)
White Tartar 4 (completely susceptible)
Appler 4 (completely susceptible)
Sterisel 4 (completely susceptible)
Belar 4 (completely susceptible)
Bond 4 (completely susceptible)
Glabrota 0 (highly resistant)

With the exception of Glabrota, all differential varieties including Bond and Victoria are susceptible in the seedling stage under greenhouse conditions. Under natural conditions the reactions of these varieties to race 101, which was introduced into a nursery by means of hypodermic injections into young shoots, were partly obscured in 1950 by the prevalence of race 45 and its allied races, 34, 57, and 68. These races appeared as a natural epidemic. Under these conditions apparently all Bond derivatives showed a completely susceptible reaction while Victoria derivatives in both seedling and adult plants showed an intermediate reaction although some, such as Victorgrain 6 and Fulgrain 4, were completely susceptible to race 101 while showing resistance to race 45 and its allies.

The reaction of Victoria and its derivatives is varied and somewhat confusing under natural as well as greenhouse conditions. Individual pustules are usually relatively large, of a full 3 to 4 type by the tenth day after inoculation, with some chlorosis surrounding the pustules and little or no necrosis. By the fourteenth day 30 to 50 percent of the pustules are surrounded by necrotic areas. However, even in those lesions where necrosis develops, the pustules are usually larger than in race 45, and secondary uredia are occasionally produced around the borders of the necrotic areas. In other words, unlike race 45 and its allies, race 101 appears capable of perpetuating itself on various Victoria derivatives by producing urediospores in relative abundance.

On Victoria and its derivatives, race 101 develops necrosis more slowly with a consequent greater susceptibility measured in terms of rust reproduction and subsequent numbers of pustules produced than for race 45. Thus at a temperature of 70°, race 45 produces necrotic lesions on rapidly growing seedlings of Victoria and its derivatives in about 8 to 10 days while race 101 produces such lesions in 11 to 14 days. Furthermore, while race 45 nearly always produces necrosis on these oats, race 101 produces such lesions as only half or less of the number of infections and there is even the possibility that purification by means of single-spore inoculations would show race 101 to include biotypes that produce no necrosis on these oats.

Of 58 collections made in nearly all the important oat-growing counties in Arkansas in 1950, 51 were found to include this new race, although race 45 and its allies predominated in nearly all of the 58 collections. Many oats volunteered and became severely infected with crown rust

during the wet summer of 1950 in this State. The rust apparently spread to early fall-planted oats from these infected volunteer plants so that these in turn were heavily infected, the severity of infection involving approximately 80 percent of the leaf area by the end of October.

At the University Farm, Fayetteville, all early fall-planted Bond derivatives except one (R19-53-4-3), all Victoria derivatives, all Red Rustproof strains, and all other winter oats such as Lee, Fulwin, Forkedeer, and Fulghum (C. I. 708) were so badly rusted that they failed to survive the rather severe winter of 1950-51. Race 101 as well as race 45 was isolated from these oats. The same varieties planted later developed very little rust because of dry fall weather and survived much better than those sown early. Race 101 was also found in an early fall-sown field of Red Rustproof and in several fields of Traveler that were severely rusted by the last week of October in Sebastian County.

The mean type of reaction of race 101 on a new set of differentials, tentatively being considered as a substitute for the older set, is as follows:

Anthony 4 (completely susceptible)	Santa Fe 1 (highly resistant)
Victoria 3 (susceptible)	Ukraine 3 (susceptible)
Appler 4 (completely susceptible)	Trispernia (highly resistant)
Bond 4 (completely susceptible)	Bondvic 2-3 (resistant to intermediate)
Landhafer 2 (resistant)	Saia 0 (highly resistant)

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RHIZOCTONIA BLIGHT OF FORAGE LEGUMES AND GRASSES[1]

J. Lewis Allison[2]

Rhizoctonia solani Kühn, the mycelial stage of Pellicularia filamentosa (Pat.) Rogers, has been known for years as a worldwide, soil-inhabiting fungus pathogenic upon many dicotyledenous and some monocotyledenous species of plants. Walker (13) groups symptoms of the disease into four phases: damping-off, stem canker and root rot, storage-organ decay, and foliage blight. The first three are well recognized but the fourth, foliage blight, is relatively uncommon. Brown patch of turf grasses is the one foliage blight commonly recognized and only a few other references are found in the literature (4, 5, 6, 7, 8, 10, 11, 12, 14).

In 1949 Rhizoctonia solani was first reported causing a foliage blight disease on certain forage legumes and grasses (3). Other reports followed (1, 9). In North Carolina, R. solani causes a destructive foliage blight disease on trefoil species (Lotus uliginosus and L. corniculatus), alfalfa (Medicago sativa) and tall fescue (Festuca arundinacea). Ladino clover (Trifolium repens), annual lespedeza species (Lespedeza stipulacea and L. striata), soybean (Glycine max), and orchard grass (Dactylis glomerata) are less seriously damaged by the disease.

Rhizoctonia blight is initiated from sclerotia of the fungus which are omnipresent in the soil. During prolonged wet hot periods sclerotia germinate, producing mycelial strands that rapidly overrun the ground and advance up onto the foliage of plants. Leaves and petioles of legume hosts become infected and water-soaked areas appear. Soon the entire leaflet, leaf, or branch collapses, and when dry these characteristically adhere to the coarser stems. The fungus advances from plant to plant wherever foliar parts touch and areas many feet in diameter may be completely blighted. Leaf blades of grass hosts become infected and irregular water-soaked areas appear. Frequently the entire blade is girdled causing a rapid collapse of all leaf area above the lesion. In dense growth all the foliage becomes blighted and settles into a water-soaked mat which becomes brittle and light in color when dry. Legume and grass plants are killed when conditions favoring the disease are prolonged, as all regenerative buds and shoots are blighted until none remain. Alfalfa plants are commonly destroyed by this means. When roots of such plants are removed from the soil and examined they are found to be in a firm, sound condition.

Rhizoctonia blight is checked during short drought periods when the soil surface and plant foliage becomes dry. When legume crops are cut for hay and grass-legume pastures are grazed or clipped before foliage growth becomes dense, sunlight and air drainage remove free moisture preventing the development of blight. In fields and pastures which are improperly managed, or when prolonged wet weather prevents harvesting or proper grazing, the foliage becomes heavy and lodges badly. Under such conditions blight is very destructive. Soybeans escape serious damage from blight, as they are usually planted in widely spaced rows and environmental conditions favoring the development and spread of the disease are lacking.

Experimental Results

Isolations were made on PDA by plating sclerotia picked from blighted plant parts and by tissue platings from infected foliage. Many isolates were obtained from trefoil, alfalfa, and tall fescue from different locations. Isolates were also obtained from Ladino clover, lespedeza, soybean, and orchard grass. Differences in cultural characteristics were apparent among isolates. There was no consistent type among isolates from the same host and similar types were obtained from different hosts, e.g., isolates from trefoil were not all alike and similiar isolates were obtained from trefoil, alfalfa, tall fescue, etc. The optimum temperature for growth of all isolates on PDA was 25° to 30° C.

Pathogenicity of each isolate was determined using the method reported by Allison and Hanson (2), both on the host from which the isolate was obtained and by cross inoculation to other hosts. Free moisture on the foliage of plants was necessary for initial infection of the fungus and subsequent development of the disease. When the foliage was allowed to dry the disease was checked with no further development until the foliage was moistened again. Optimum temperature for disease development was 25° to 30° C. Penetration by hyphal strands was both through the cuticle and stomata.

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A range in virulence from high to low was obtained among isolates from a single host when tested on this host, e.g., isolates from trefoil ranged from high to low on trefoil. Isolates high or low in virulence on one host reacted in a like manner on other hosts, e.g., isolates from trefoil reacted in a like manner on alfalfa, tall fescue, and soybean. Isolates of high virulence causing severe foliage blight were equally virulent in causing damping-off when seed of each host being tested were immersed in a mycelial suspension and planted in sterile soil. Isolates of low virulence, as measured by incidence of foliage blight, caused no damping-off in comparable tests.

On the basis of the results obtained the legume and grass species tested were rated in respect to their susceptibility to Rhizoctonia foliage blight: (1) Very susceptible -- Lotus uliginosus, L. corniculatus and Festuca arundinacea. (2) Susceptible -- Medicago sativa, Lespedeza stipulacea, L. striata, and Glycine max. (3) Resistant -- Trifolium repens and Dactylis glomerata.

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NORTH CAROLINA AGRICULTURAL EXPERIMENT STATION AND THE DIVISION OF FORAGE CROPS AND DISEASES, U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

A PRELIMINARY REPORT ON ETIOLOGICAL AND CONTROL STUDIES OF DAMPING-OFF AND ROOT-ROT IN THE CONIFER SEEDBED IN DELAWARE[1]

R. S. Cox[2]

INTRODUCTION

The conifer seedbeds of the State Forest Tree Nursery, Lincoln, Delaware, were found in November, 1949, to be in a seriously diseased condition. The local name of the disease, "three-year root rot", was derived, primarily, from symptom-expression on three-year-old white pine (Pinus strobus L.) seedlings, although seedlings of Norway spruce (Picea abies (L.) Karst.), and Douglas fir (Pseudotsuga taxifolia caesia (Schwer.) Aschers. & Graebn.) were also affected. The disease is characterized, on three-year-old white pine, by severe browning and casting of the needles. Symptoms are first evidenced in August by a browning of the needle tips. This is followed rapidly by a necrotic condition extending the entire length of the needles. The transformation from green, apparently-healthy needles to severe defoliation has been known to take place within a two-week period.

Examinations revealed that the disease was not restricted to three-year-old seedlings, but that seedlings of all three species were susceptible during all stages of growth in the seedbed, up to three years. Loblolly pine (Pinus taeda L.) appeared not to be greatly affected.

A high degree of mortality occurred among seedlings of the three susceptible species throughout the first year and, to a lesser extent, during the second and third years. Frequently, this was followed by needle-cast and death of white-pine seedlings in fall of the third year as described above.

It is well established that several fungi are capable of causing "damping-off", "late damping-off", and "root-rot" of conifer seedlings (1). Also, it has been suggested recently that nematodes may be an important factor in a root-rot of longleaf pine (P. palustris Mill.) and slash pine (P. caribaea Morel.) in Mississippi (3). It appeared questionable, however, that the action of damping-off fungi and/or nematodes could account adequately for the sudden browning and shedding of needles of white-pine seedlings in fall of the third year; neither did the so-called "needle-cast" fungi appear to be of major consideration, because fruiting bodies of these organisms were not found.

Since Lincoln, Delaware, is situated on the southern fringe of the Coastal Plains white-pine range, and since the State Forestry Nursery is located on very sandy soil, the prolonged action of fluctuating temperatures and rainfall in winter and summer, possibly aggravated by a limited root system due to root rot, appeared to be a possible contributory factor to the three-year phase of the disease. Conifer seedlings are known to be susceptible to excessive drought and extreme temperatures (2).

This paper is a preliminary report on the results of: (a) isolations from stems and roots of variously-aged seedlings of white pine, Norway spruce, and Douglas fir, and subsequent greenhouse pathogenicity studies; (b) the effect of several soil fumigants on stand- and disease-development; and (c) the effect of laths as partial shade.

ETIOLOGY

Isolation data are summarized in Table 1. Several fungi were consistently isolated in culture from diseased stems and roots. One form closely fitted the description of Rhizoctonia solani Kühn and will be so referred to in this paper. Other forms were tentatively considered as species of Fusarium, Phoma, Botrytis, and Trichoderma. Still another form, as yet unclassified, resembled R. solani in respect to mycelial branching, septation and sterility, but did not exhibit macroscopic features of that species. Trichoderma predominated in cultures where surface-sterilizing agents were not employed. Since this fungus is generally considered as a common soil-saprophyte, it was not included in pathogenicity studies.

It is interesting to note that, except from Douglas fir, Rhizoctonia was isolated more

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[2]The field work was made possible through the cooperation of W. S. Taber, State Forester, and other members of the Delaware State Forestry Department. The assistance of D. J. Morton and G. B. Armstrong, senior students, University of Delaware, during certain phases of this work is gratefully acknowledged.

Table 1. Results of isolations from stems and roots of conifer seedlings from State Forestry Nursery, Lincoln, Delaware. [a]

Host plant	Age of seedlings (years)	Number of pieces plated	Fungi isolated [b]					
			Rhizoctonia	Fusarium	Trichoderma	Phoma	Botrytis	Sterile form
White pine	1/2 or less	262	83	52	30	1	2	6
	1	55	5	14	0	0	10	0
	2	74	14	32	46	0	15	1
	3	338	40	191	61	0	10	0
Total		729	142	289	137	1	37	7
Norway spruce	1/2 or less	392	217	39	8	76	5	40
	2	62	19	25	1	0	10	1
Total		454	236	64	9	76	15	41
Douglas fir	1/2 or less	132	17	43	15	0	16	8
Grand Total		1315	395	396	161	77	68	56

[a] Four isolation techniques were used: (1) diseased pieces dipped in 20% Clorox for 3 minutes and plated directly; (2) dipped in bichloride of mercury (1-1000) 1/2 minute, washed in 3 changes of sterile distilled water and plated; (3) washed in 3 changes of sterile distilled water and plated; and, (4) washed in sterile water and planted in apples from which cores approximately 3/4 inch depth had been removed by means of a flamed cork-borer 3 mm. in diameter.

[b] Number of isolates of each organism from specified seedlings.

frequently than other fungi from seedlings six months old or less, whereas on older seedlings (isolations primarily from rootlet lesions) Fusarium predominated.

Greenhouse pathogenicity tests were undertaken to determine, if possible, the relative importance of these organisms in respect to pre- and post-emergence damping-off and to root rot.

In one test, using seven one-week-old single hyphal tip fungous isolates grown on potato-dextrose agar for inoculum, 128 pots containing steamed soil were divided into four equal lots of 32 pots each. Each lot of pots was seeded (100 seed per pot) to one of the four species: white pine, Norway spruce, Douglas fir, and loblolly pine. [3] Four pots of each lot of 32 were inoculated with one of the seven fungous isolates, one petri plate culture per pot, leaving four pots of each lot untreated to serve as checks. The fungous cultures, disced up into approximately one-quarter inch squares, were placed directly over the uncovered seed. The seed and inoculum were then covered with a layer of steamed soil approximately one-quarter inch in thickness. The test ran from February 23 to April 17, 1951. Starting on March 9, stand counts were made three times weekly. The number of dead seedlings was recorded and discarded (or used for subsequent isolations) each time a count was made. At the end of the test, roots were examined for root-rot symptoms. Results on stand and post-emergence damping-off are recorded in Table 2.

These data leave many questions unanswered, but the following points of interest are noted:

(a) All three isolates of Rhizoctonia presumably caused pre-emergence damping-off of Norway spruce and white pine, since total emergence (healthy plus dead seedlings) was considerably less than that of the checks.

(b) All Rhizoctonia isolates caused post-emergence damping-off of Norway spruce, white pine, loblolly pine, and probably Douglas fir. The fungus was recovered in culture from a high percentage of the damped-off seedlings in these Rhizoctonia series.

(c) The sterile form showed a slight degree of pathogenicity on Norway spruce and

[3] Seed of white pine, Douglas fir and Norway spruce were stratified in moist sand at 40° F. for one month immediately prior to planting.

Table 2. Total emergence and percent post-emergence damping-off of conifer seedlings 50 days after seeding in steamed soil artificially inoculated with various fungus isolates in the greenhouse.

Isolates [a]	Host Plants							
	Norway spruce		White pine		Loblolly pine		Douglas fir	
	Total [b] emergence (Number)	Post-emergence damping-off (percent)	Total [b] emergence (Number)	Post-emergence damping-off (percent)	Total [b] emergence (Number)	Post-emergence damping-off (percent)	Total [b] emergence (Number)	Post-emergence damping-off (percent)
Check	247	1	122	0	258	1	44	38
R-1	79	62	68	22	231	11	40	50
R-2	50	52	49	37	203	33	55	58
R-3	85	51	81	35	215	27	14	43
St. F.	223	17	93	2	193	16	50	48
Phoma	243	7	125	1	247	3	30	27
Fusarium	244	3	104	0	216	0	18	72
Botrytis	188	8	114	2	220	1	38	6

[a] The fungus isolates are identified as follows:

R-1, Rhizoctonia solani isolated from Douglas fir rootlets
R-2, Rhizoctonia solani isolated from Norway spruce rootlets
R-3, Rhizoctonia solani isolated from white pine stems
St. F., sterile form isolated from Norway spruce rootlets
Phoma isolated from Norway spruce rootlets
Fusarium isolated from white pine rootlets
Botrytis isolated from white pine rootlets

[b] From a total of 400 seed planted.

and loblolly pine. It was successfully recovered in culture from diseased seedlings of these two species.

(d) The Fusarium, Phoma, and Botrytis isolates appeared to be, at best, only very mildly pathogenic. Of these three isolates, Fusarium was recovered in culture from diseased Norway spruce and white pine seedlings, none was recovered from loblolly pine and Douglas fir. This is not considered as final proof of the pathogenicity of Fusarium, since this fungus was occasionally recovered from dead seedlings in other series.

(e) Due to poor germination, about 10 percent, and subsequent dying of many seedlings in each series, definite conclusions could not be drawn in the case of Douglas fir. It appeared likely, however, that the Rhizoctonia isolates were pathogenic on Douglas fir, since they were consistently recovered in culture from diseased seedlings in the R-1, R-2, and R-3 series. In the case of Fusarium, although 72 percent of the seedlings died, the fungus was not re-isolated.

(f) Only the Rhizoctonia isolates caused typical root-rot symptoms (data not shown in Table 2). Roots of loblolly pine were not noticeably affected.

CONTROL

A field experiment was set up April 5, 1950, to study the effect of several soil fumigants on control of the various stages of the disease. Standard nursery beds were divided into 4 x 5 foot plots, each plot receiving one of seven treatments (including a check). The randomized block system was employed with two replications for each treatment. Each plot was divided equally, lengthwise; and one-half was sown to white pine and the other to Norway spruce, both at standard seeding-rates. Data on stands (apparently healthy seedlings) and seedling heights taken May 17, 1951, are shown in Table 3.

Table 3. Effect of various soil fumigants on subsequent stand and height of conifer seedlings approximately one year after seeding. [a]

Treatment [b]	Rate of application gals./acre	White pine		Norway spruce	
		Stand [c]	Height [d] (mm)	Stand [c]	Height [d] (mm)
Check	--	34	87	26	76
Chloropicrin	40	125	185	58	145
Formaldehyde (40%)	435	101	128	43	136
Dichloropropene mixture	28	48	82	20	103
	57	47	101	43	114
Ethylene dibromide	28	27	80	26	103
	57	54	95	26	110
L.S.D. .05		22		N.S.	

[a] Notes relative to soil treatments: soil treated April 5, 1950. Chloropicrin confined with water seal. Formaldehyde applied as a drench (1 50) at rate of 1/2 gal./sq. ft. and confined with roofing paper for 5 days. Remaining materials (including chloropicrin) applied with hand injector. Soil temperature at time of fumigation 55° F.

[b] The following materials were furnished by the manufacturer: Chloropicrin (Larvacide) and ethylene dibromide (Iscobrome D) by Innis-Speiden Company; Formaldehyde by E. I. duPont deNemours & Co.; dichloropropane-dichloropropene mixtures (D-D) by Shell Chemical Company.

[c] Average of 2 samples taken from a sq. ft. area in each plot.

[d] Average of 10 seedlings.

Definite conclusions cannot be drawn at this early date relative to the effectiveness of the various materials against all stages of the disease, but it appears that some definite trends are being established. Chloropicrin appears most promising from the standpoint of both stand and seedling-height. Formaldehyde, also, shows a considerable degree of promise. The beneficial effect of these two materials has apparently lasted throughout the first year. Dichloropropene mixture and ethylene dibromide apparently do not give adequate control. Since these two materials are primarily nematocidal in action, the data would tend to eliminate the action of nematodes as a contributory factor to the damping-off phases of the disease. This soil-treatment-program is being expanded in 1951.

To check on the possibility that the browning of needles and death of three-year-old, white-pine seedlings was due, in part, to the prolonged action of summer and winter injuries, one bed of two-year-old white-pine seedlings was covered with laths during July and August, 1950, and again during the four-month period from December 1, 1950, through March 31, 1951.[4] The bed was compared on August 31, 1950, and again on March 31, 1951, with an adjacent uncovered bed. Plants in the "covered" bed exhibited, in general, a much greener foliage than those in the "uncovered" bed on both dates. Also, necrosis of the needle tips was less pronounced in the bed which had been covered.

Another interesting observation was made on May 25, 1951. Growth of new shoots in the "covered" bed was proceeding at about twice the rate as that in the "uncovered" bed. This difference in growth-rate resulted in a marked difference in average seedling-height of the two beds. This phenomenon appears to be associated with the effect of the laths in reducing the intensity of direct sunlight reaching the plants, thus preventing extreme discrepancies between water-absorption and transpiration. The cumulative effect of laths in summer and winter would appear to be that of a reduction of needle-necrosis due to desiccation. Thus, in turn, would ensure a more vigorous plant.

[4] The laths were arranged so that the plant beds received approximately 50 percent of the available sunlight.

It is interesting to speculate on the value of proper soil-treatment, coupled with adequate precautions against heat, cold, and drought-injury to the seedlings, as a control program for the disease-complex during the three-year period of growth in the seedbed. This possibility is being investigated.

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THE VIABILITY OF SPORES AND MYCELIUM OF ENDOCONIDIOPHORA VIRESCENS ON SUGAR MAPLE LUMBER

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The sapstreak disease of sugar maple[2], caused by the fungus Endoconidiophora virescens, results in grayish to reddish areas in the sapwood of infected trees. E. virescens, in addition to causing sapstreak, is a common cause of sapstain in hardwood lumber throughout the East. Tests are now being conducted to determine whether isolates from different sources are similar with respect to pathogenicity. Large merchantable trees are killed by the disease. The causal fungus is readily isolated from the discolored areas in standing trees or in freshly cut lumber. After infected trees are cut, the fungus fruits abundantly on the exposed discolored areas of the logs or on the sawn lumber, causing extensive black discolorations. So far as is known, the sapstreak disease on sugar maple is confined to the Big Ivy section of the Pisgah National Forest, near Barnardsville, North Carolina. Isolated cases of a similar disease have been found on yellow-poplar in northeastern Tennessee and near Asheville, North Carolina.

Most of the sugar maple lumber cut in the affected area is used locally. Occasionally, shipments are made outside the area, perhaps even into other maple-producing areas. It was questioned whether the surface fruiting of the fungus already in the logs when the boards are sawn might spread the fungus to other areas. The purpose of this study was to find out the length of time spores and mycelium of Endoconidiophora virescens would remain viable in or on cut lumber, with a number of the more common storage methods, including spraying with a sapstain preventive.

In June 1949, ten logs were selected from recently cut sapstreaked trees. Wood cultures were made from near the ends of several of the logs and Endoconidiophora virescens was easily isolated from the discolored areas.

The logs were then cut into 1-inch boards of varying widths and from 6 to 10 feet in length. Twenty boards were used in each of six treatments, or 120 boards in the experiment. As the boards were removed from the saw, cultures were attempted from every fourth board. All wood cultures were made by sawing a section from the board and culturing from the center of the split section. As soon as the cultures were made, 40 of the boards were sprayed with a solution of Permatox 10-S[3] at the rate of 10 pounds per 100 gallons of water. Twenty of the treated boards were bulk-piled and 20 were sticker-piled. The remaining 80 untreated boards were handled in the following ways: 20 boards bulk-piled green; 20 boards sticker-piled green; 20 boards end-racked green; and 20 boards bulk-piled for two weeks, then kiln-dried. With the latter, spore cultures were attempted immediately before, and spore and wood cultures immediately after kiln-drying. Spore or wood cultures were attempted from five boards in each treatment one, two, and three months after the boards were sawed.

Results

Positive cultures of Endoconidiophora virescens were obtained from 73 percent of the cultures attempted from the freshly cut boards. For the kiln-dried boards, positive cultures were obtained from 100 percent of the spore cultures attempted immediately before drying (Table 1). After kiln-drying, the fungus was not obtained from either spore or wood platings.

Boards treated with Permatox 10-S did not yield positive cultures from either spore scrape or wood cultures, at any time after treatment. These boards remained bright and free of obvious surface fungus growth throughout the experiment, as compared with the dense black mycelial growth on the untreated air-dried boards (Fig. 1). Vapors from these treated boards prevented fungus growth on several layers of boards used in covering the pile. There was no apparent difference in subsequent staining between the bulk-piled and sticker-piled treated boards.

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[1]Associate Pathologist, Division of Forest Pathology, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture, in cooperation with the Southeastern Forest Experiment Station.

[2]Hepting, George H. 1944. Sapstreak, a new killing disease of sugar maple. Phytopath. 34: 1069-1076.

[3]Active ingredients - Borax and Sodium Pentachlorophenate, manufactured by Chapman Chemical Company, Memphis, Tennessee.

Table 1. Recovery of Endoconidiophora virescens from lumber cut from sapstreaked trees. [a]

Treatment	Boards yielding positive Endoconidiophora virescens isolations (1949)							
	Prior to treatment		After treatment					
	June 15[b]	June 27	July 8		August 11		September 9	
	Wood	Spore	Wood	Spore	Wood	Spore	Wood	Spore
	%	%	%	%	%	%	%	%
Permatox 10-S								
Bulk-piled	80	-	-	0	0	-	0	-
Sticker-piled	60	-	-	0	0	-	0	-
Piled green								
Bulk-piled	80	-	-	60	-	0	0	0
Sticker-piled	80	-	-	60	-	0	0	0
End-racked	60	-	-	40	-	0	0	0
Bulk-piled, then kiln-dried on June 27	80	100	0	0	0	-	0	0

[a] Each treatment represented by 20 boards, and each period 5 boards were sampled from each treatment.

[b] Boards fresh from the saw and before treating or piling.

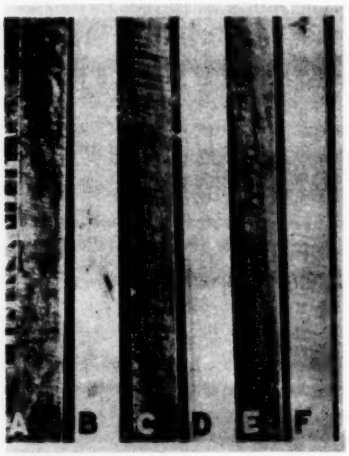

FIGURE 1. Comparison of green-bulk-piled sapstreaked lumber (A, C, and E) and lumber from same logs sprayed with Permatox 10-S ten days after sawing (B, D, and F). Note absence of fungus on treated boards, even though it was present in the wood.

The end-racked boards yielded positive spore-scrape cultures from 40 percent of the boards sampled at the end of one month. At the end of two and three months no positive cultures were obtained from either spore scrapes or wood platings. Most of the fungus growth on the end-racked boards was on the end nearest the ground, the other end being relatively free of subsequent fungus growth.

At the end of one month the boards that were either bulk-piled or sticker-piled green yielded 60 percent positive cultures from spore isolations. Neither of these treatments yielded the fungus from spore cultures at the end of two months, or from spore or wood cultures at the end of three months. All of the boards in these two treatments were heavily covered with black mycelial growth of Endoconidiophora virescens soon after the experiment started.

Conclusions

Lumber piling methods that might be used by southern sawmills were employed to determine the length of time spores and mycelium would remain viable in lumber cut from trees infected with Endoconidiophora virescens. Spraying the boards with Permatox 10-S prevented the development of spores and mycelium on the surface of the infected boards. Kiln-drying killed both the spores and mycelium. However, the time that elapsed between cutting and kiln-drying in these tests permitted the development of heavy discoloration, due to the fungus, on the exterior of the boards.

End-racking, while not preventing the development of external stain, greatly reduced it, the stain being confined to those portions of the boards closest to the ground.

Bulk-piling and sticker-piling of green boards permitted entire boards to become heavily stained.

Spores and mycelium of the fungus Endoconidiophora virescens did not remain viable as long as two summer months after the logs were cut into lumber, regardless of how the lumber was

treated. These results agree closely with those of Scheffer and Chidester[4] who found that E. virescens died in less than one month in laboratory-infected dry pine.

Adequately seasoned or fungicide-treated sapstreak lumber can be safely shipped into other sugar-maple-producing areas with little danger of spreading the disease.

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[4]Scheffer, Theodore C., and Mae S. Chidester. 1948. Survival of decay and blue-stain fungi in air-dry wood. Southern Lumberman 177 (2225): 110-112.

OAK WILT FOUND IN WEST VIRGINIA

R. P. True, F. W. Craig, and H. L. Barnett [1]

Oak wilt, caused by Chalara quercina, has been found in West Virginia near Morgantown in Monongalia County, near Huntington in Cabell County, and near Riverton in Pendleton County.

These three nearly simultaneous discoveries of oak wilt in widely separated counties [2] are the first fruits of an informal statewide survey being participated in by personnel of the West Virginia Department of Plant Pathology and Bacteriology, the West Virginia Conservation Commission, and the West Virginia State Department of Agriculture. Cooperating Federal agencies include the U. S. Forest Service staffs of the Monongahela and George Washington National Forests, and Blister Rust men of the U. S. Bureau of Entomology and Plant Quarantine. Private foresters, arborists, and County Agricultural Extension Agents have also been of help in reporting suspected cases of oak wilt. This informal survey is being coordinated by R. P. True and F. W. Craig. The West Virginia survey is one of several inspired and informally assisted by the Division of Forest Pathology, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Department of Agriculture, in States possibly threatened by the oak wilt disease.

Trees so far found at Morgantown are in two locations about four miles apart, and in each location the disease seems to have been present for at least one year previously. One owner reports losing 12 trees near his house during the last eight or nine years, all dying with symptoms similar to the oak wilt-infected trees found this year in his yard.

Trees found near Huntington are at a single spot where the owner reports losing five oak trees from his yard with oak wilt symptoms during the last five years.

Near Riverton the two trees first found affected with oak wilt seem to have been affected this year for the first time. A dead oak with adjacent symptom trees was found about a mile distant. Trees near the dead tree have now been proved by culturing also to be affected by oak wilt.

There is a strong suggestion that oak wilt has been present though unrecognized in West Virginia for several years. It appears to be rather widespread. The purpose of the survey is to determine how generally prevalent the disease is over the State, to learn more about the length of time it may have been present, and to determine how much serious damage it may have done during that period and seems likely to do in the next few years.

Oak wilt appears to be spreading in West Virginia, not only by root grafts but also by other agencies capable of carrying the disease to more distant trees.

There is no immediate evidence that the disease is now so severe in the locations found as to threaten the early loss of any considerable volume of timber. There is no present indication of any need to sell oak timber at small diameters or at salvage prices in West Virginia.

Decision regarding the adoption of a State-wide program of eradication will not be made until the results of the survey give a clear and more complete basis for such decision. The immediate undertaking of attempted eradication seems unwise. [3]

WEST VIRGINIA UNIVERSITY AND WEST VIRGINIA STATE DEPARTMENT OF AGRICULTURE

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[1] Respectively: Associate Plant Pathologist, West Virginia University; Entomologist, West Virginia State Department of Agriculture; and Mycologist, West Virginia University.

[2] Morgantown oak wilt trees were identified as such from samples collected by Barnett and True. The fungus was sent to T. W. Bretz of the Division of Forest Pathology at Columbia, Missouri, for confirmation of Barnett's identification. All subsequent determinations have been made by Barnett. The tree near Huntington was found on the basis of samples sent in independently by W. D. Click, Agricultural Extension Agent, and Edward Thomas, Farm Forester with the State Conservation Commission. Samples from trees near Riverton were submitted by Henry Yost and C. Fultz of the White Pine Blister Rust Agency of the Bureau of Entomology and Plant Quarantine, U. S. Department of Agriculture.

[3] Latest findings show that the disease also is present in Boone and Wood Counties in addition to the locations mentioned above.

SOME FURTHER REPORTS ON OAK WILT

OAK WILT FOUND IN MICHIGAN

By Forrest C. Strong

The oak wilt disease caused by Chalara quercina was found in a red oak tree located in Cass County, Michigan. The nearly dead tree was found by Louis Miller, Farm Forester, State Conservation Department. Isolation of the causal fungus was made by the author and confirmation of its identity was made by Dr. T. W. Bretz, Division of Forest Pathology, U. S. Department of Agriculture. While the presence of the disease has been suspected for some time, this is the first authentic case in this State.

Plans are completed to make an aerial survey of the two southern tiers of counties and a few other counties, beginning the second week in July. Samples from suspected trees will be collected for laboratory diagnosis. This work will supplement a study of all cases of dying of oak trees, which has been carried on since 1945.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, MICHIGAN STATE COLLEGE, EAST LANSING

OAK WILT IN WESTERN PENNSYLVANIA

By F. R. Lancaster and A. F. Rumph

The oak wilt disease fungus, Chalara quercina Henry, was isolated from three trees on one property in Sewickley Heights, Allegheny County, Pennsylvania. Collections from these trees were made May 31, 1951. The trees are all pin oaks (Quercus palustris).

It is believed that this is the first report of the occurrence of oak wilt in western Pennsylvania. In August of 1950, we were concerned that these might be oak wilt trees. However, we were unable to verify our suspicions. Cultures were all negative. There was rapid wilting, defoliation and dieback rather evenly distributed over the tree crowns both in 1950 and 1951. Of course, a great deal more of the crowns of the trees are affected this year over last. There is much clump sucker growth now. No leaf bronzing was noticed in either year.

In 1950, we could not find sapwood streaking of the type described for oak wilt. In 1951, we found streaks with almost every cut.

THE DAVEY TREE EXPERT COMPANY

OAK LEAF BLISTER SEVERE IN SOUTH MISSISSIPPI

Berch W. Henry

Leaf blister, caused by the fungus Taphrina caerulescens, is endemic on oaks in the Southern States. It varies in severity from year to year, and between species and individual trees of a species in any given year.

Infection reportedly occurs only on immature foliage, largely that developing in early spring, and is favored by cool, wet weather. [1] Following infection, an oval or circular pocket is formed on the leaf blade, which causes a bulge or blister on the opposite surface. There may be several blisters on one leaf. Many infected leaves are soon shed, and the tree tends to become weakened and its growth rate reduced by the premature and sometimes heavy defoliation. The disease can reportedly be controlled by the application of Puratized Agricultural spray plus ziram before the buds open in the spring. [2]

A large number of requests were received during May 1951 from property owners in south Mississippi for information concerning their diseased oak trees. In every case, oak leaf blister was the disease and the resulting discolored foliage and excessive defoliation had caused the concern of the owner. Water oak (Quercus nigra), willow oak (Q. phellos), laurel oak (Q. laurifolia) and live oak (Q. virginiana) were found diseased, but water oak was the only species found severely defoliated. In individual cases at Hattiesburg and Purvis, approximately 50-60 percent of the foliage had been shed by the end of May. Trees less severely damaged were putting on new growth but no new growth was found on those severely defoliated. Though these particular cases involved only shade and ornamental trees, the disease was present on forest trees in the vicinity.

In south Mississippi, the temperature was below normal and the rainfall above normal for the month of April while the reverse held for the month of May. Thus the cool, wet weather of April could have favored the build-up of the disease and the host, dry weather of May probably encouraged defoliation of the diseased leaves while retarding the development of new ones.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE

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[1] Weber, George F. Leaf blister of oaks. Florida Agr. Expt. Sta. Press Bul. 558. 1941.
[2] Hepting, George H. Leaf blister of oak controlled by spraying. Arborist's News 16(6): 53-55. Illus. June 1951.

NOTES ON OCCURRENCE OF SOME PLANT DISEASES IN MARYLAND DURING 1950

W. F. Jeffers, C. E. Cox, R. A. Jehle,
L. O. Weaver, O. D. Morgan, and J. E. Moore

During 1950 several plant diseases, not previously reported in Maryland or of infrequent occurrence, were observed. The various members of the Plant Pathology Division at the University of Maryland have combined their observations in the following list.

1. Phytophthora cinnamomi was found to be responsible for serious losses to woody ornamental plants in the nursery and in home plantings. Azalea, Japanese holly, juniper, rhododendron, viburnum, and yew were affected. As usual this organism caused most loss in heavy, poorly-drained soil. However, in one planting of viburnum the disease was rather uniformly distributed over a well-drained area. P. cinnamomi was isolated from several of the different plants mentioned above and in all cases there were dark streaks extending up the crown and lower stem. This dark discoloration is said to be typical of Phytophthora crown and root rot.

2. Thelephora spiculosa was observed in a home garden near College Park, where it apparently killed several ornamental plants including fern and azalea. The fungus forms a dense weft of mycelium on the surface of the soil and on plants. This organism was identified by Mr. P. L. Lentz of the Division of Mycology and Disease Survey, U. S. Department of Agriculture.

3 An unknown trouble was serious on dogwood in suburban areas near Washington, D. C. The general symptoms were very similar to injury by 2, 4-D but the trouble was so widespread that this cause had to be eliminated. Also, there would often be several healthy trees adjoining affected trees. Leaves were misshapen, stunted, and light colored; they turned red early and often dropped prematurely. Numerous fungi were isolated from affected leaves but none were proved to be pathogenic to dogwood.

4. Botrytis infection of dogwood leaves resulted in many cases when fallen flower bracts adhered to the leaves. At the point of adherence Botrytis was often able to become established and cause death of more of the leaf tissue.

5. Elsinoe corni was present on dogwood blossoms, fruit, and foliage at several locations in southern Maryland. Spot anthracnose is not common in this area but occasionally causes much disfigurement of the flower bracts. The disease seems to occur along roadsides rather than in wooded areas.

6. Several varieties of ornamental rhododendrons were observed to have a virus-like, yellow mottling of the foliage. Tests are being conducted to determine possible graft transmission of this condition.

7. Ovulinia azaleae, cause of azalea flower blight, occurred in a few azaleae plantings. This disease has been known to be present in Maryland for several years but so far does not seem to be spreading very rapidly. However, it is considered that flower blight constitutes a potentially serious threat to azaleas in the vicinity of Washington, D. C.

8. Fusarium roseum was isolated from chrysanthemum buds affected with a serious rot that destroyed the unopened flower. When pure cultures of this organism were used for artificial inoculation of chrysanthemum buds the same symptoms were produced. This disease was observed in two greenhouse plantings.

9. Several diseases of orchid were observed in standard commercial types. Bacterial leaf spot is quite common on several genera and sometimes causes serious damage. A ring-spot type of lesion that appears to be of virus nature was occasionally found. A species of Colletotrichum commonly developed on weakened orchid leaves.

10. Macrophoma fici was found to be associated with a canker disease of cultivated fig, probably Ficus carica. This is believed to be the first report of this organism from Maryland.

11. Sphaceloma viburni caused serious damage to foliage of Viburnum opulus at two locations in Somerset County. This organism had not previously been reported from Maryland.

12. Sphaceloma hederae was found on English ivy growing near Salisbury. This was likewise the first record of this organism in Maryland.

13. Dutch elm disease (Ceratostomella ulmi) killed a number of elms in Montgomery County near Washington, D. C. A number of these trees showed the acute stage which results in complete death of the tree within about ten days. Such trees presumably were affected the previous summer but showed few symptoms at that time. Nearly all of the cambium in the large branches, main trunk, and upper root area was discolored in cases where such rapid death occurred.

14. A virus disease of tobacco resembling leaf curl was found for the first time in Charles County, Maryland. The disease has been graft-transmitted to tomato variety Marglobe and to Datura stramonium.

15. Black shank disease of tobacco was first found in Anne Arundel County, Maryland, in 1949 on a half-acre field of Maryland Broadleaf. The organism (Phytophthora parasitica var. nicotianae) was again found in 1950 in three fields in Charles County.

16. Hollow stalk, a bacterial disease (Bacillus [Erwinia] aroideae) rarely found in Maryland, caused some damage in a 15-acre field of tobacco. The disease apparently came from a nearby patch of rotten cabbage. The grower had topped his tobacco with the same knives used to cut the cabbage.

17. Nigrospora cob rot was found on field corn for the first time in Maryland. The fungus (Nigrospora ozyzae) was found on a small number of ears in two fields.

18. Alfalfa rust (Uromyces striatus var. medicaginis) and clover rust (Uromyces trifolii) have been found in trace amounts in various areas of Maryland.

19. Sclerotinia trifoliorum caused considerably more damage to crimson clover than usual.

20. Sclerotinia sclerotiorum was also abundant and caused damage to such crops as turnip, lima bean, and Boston yellow daisy.

21. Stemphylium solani was recognized for the first time as a cause of serious tomato leaf injury. In localized areas on the Eastern Shore it was the principal foliage trouble.

22. Meloidogyne hapla was found to be present in Pontiac potatoes showing small, raised areas on the surface. This species of root-knot nematode attacks other crops in Maryland but this was the first definite record of its occurrence on white potato.

23. Claviceps purpurea was unusually abundant on rye in the Eastern Shore area. Heaviest infection occurred around the edges of fields. Ergot sclerotia were also found on flower stalks of orchard grass [Dactylis glomerata] in this area.

24. Sensitivity of the Jersey blueberry variety to excess soil moisture was quite evident in several locations. In one instance alternate rows of the Burlington variety were uninjured while Jersey plants were severely stunted or killed. Jersey also seems to be more responsive to other unfavorable soil conditions. This is of sufficient importance in Maryland to warrant the recommendation that Jersey should not be used for commercial planting in certain areas.

25. Blueberry stunt [virus] was found, for the first time, in Maryland. The Scammell variety was severely affected in one location.

26. The constriction canker disease [Phomopsis] of peach was observed in several locations. In one commercial planting it has caused severe damage. Golden Jubilee is quite susceptible.

27. Occurrence of another strain of the strawberry red stele organism (Phytophthora fragariae), capable of causing severe infection of Temple and Fairland under field conditions, was observed in the commercial strawberry fruit production area on the Eastern Shore.

UNIVERSITY OF MARYLAND, COLLEGE PARK, MARYLAND

TWO SPECIES OF PYTHIUM RECORDED FROM VENEZUELA

Antonio Ciccarone and John T. Middleton

Extensive losses of tobacco seedlings in the seedbeds of the "Compañia Venezolana de Tabaco" at Maracay and Turmero in the State of Aragua were reported in October during the dry season of 1947-1948. The roots of affected plants were poorly developed, stunted, and decayed. The stems were shrunken and markedly constricted at soil level. Diseased plants wilted during the hottest hours of the day and frequently collapsed. Overcrowded plant stand and poorly drained, heavy, black soil were important factors favoring disease development. A Pythium sp. was consistently isolated from the diseased roots and stems. Attempts to arrest spread of the disease by application of Bordeaux mixture to the seedbeds were unsuccessful.

A similar Pythium sp. was isolated from alfalfa seedlings from "La Providencia", Maracay, in May, at the end of the dry season mentioned above. A large experimental planting of alfalfa had been row-planted in a heavy, black, level soil. The alfalfa emerged perfectly, but soon the plants became diseased and collapsed, ultimately lying uniformly on the ground as though damaged by a strong wind. The roots of affected plants showed water-soaked lesions. The disease spread rapidly and could not be checked by improved drainage and soil aeration.

Apparently the same Pythium sp. was isolated from seedlings of frijol (Vigna sinensis), grown at "Gonzalito", Maracay. Although the soil was black and heavy, little plant loss was sustained.

The species of Pythium isolated from tobacco, alfalfa, and frijol seedlings has in every case been identified as P. aphanidermatum (Edson) Fitz. So far as the writers are aware this is the first report of the species from alfalfa.

Experimental plantings of Sorghum vulgare (milo) were established on black, heavy soil at "Gonzalito", Maracay, in March of the dry season of 1947-1948. The plantings were periodically furrow-irrigated. Some of the plant stands were irregular and an examination and isolation from diseased seedlings was made. The mesocotyls of affected plants were constricted and showed a reddish discoloration. A Pythium sp. was isolated. Affected plants seemed to withstand the disease for some time.

Many pathological phenomena are reported on rice grown in the Tachira region. Platings were made from a specimen of Tachira soil (specific origin of the sample not known) for the purpose of determining if possible what kinds of fungi were present. A Pythium sp. was isolated which appears to be identical with that secured from milo at Maracay. The importance of this fungus on rice remains unknown.

Both of the isolates from milo and from rice soil have been identified as Pythium arrhenomanes Drechs.

As far as we are able to determine this report constitutes the first record of Pythium spp. from Venezuela.

The finding of P. aphanidermatum and P. arrhenomanes in a few hosts, one of which is new, and the economic importance of some of the phytopathological phenomena induced by these fungi, seems to be good evidence that these organisms may be commonly distributed in Venezuela. Further study may afford information of some value to phytopathological investigations in Venezuela.

STAZIONE DI PATOLOGIA VEGETALE, ROME, ITALY; AND CALIFORNIA AGRICULTURAL EXPERIMENT STATION, RIVERSIDE, CALIFORNIA

NEW RECORDS OF PLANT DISEASES

NEMATODE ROOT ROT OF COTTON IN LOUISIANA

By W. J. Martin, L. D. Newsom, and Jack Schwegmann

On June 4, 1951 the writers observed a brown root rot affecting cotton plants in Bossier Parish 3 miles south of Bossier City, Louisiana. The most severely affected plants were found among stunted plants on knolls in that area. Severity of the disease ranged from small, brown spots on the smaller roots to almost complete decay of the roots. Specimens of the affected plants were brought into the laboratory for examination. Tissue isolations from the necrotic spots on the roots failed to yield fungi.

Since the root rot was strikingly similar to that described by Graham,[1] the roots were examined for nematodes according to the method described by Graham. Nematodes were found to be present in abundance and they appeared to fit descriptions of those causing Nematode Root Rot. Specimens of the nematodes were prepared and sent to Dr. Graham, who identified them as typical Pratylenchus leiocephalus.

This is the first report of the occurrence of this nematode in Louisiana.

DEPARTMENT OF PLANT PATHOLOGY AND DEPARTMENT OF ENTOMOLOGY, LOUISIANA AGRICULTURAL EXPERIMENT STATION, BATON ROUGE, LOUISIANA

A CORRECTION

Our attention has been called to an error in Table 2, page 74 of Supplement 202, under DELPHINIUM. Diplodinia delphinii was reported in Connecticut, New Jersey and New York as "A new Phoma disease of perennial delphinium." (Abstr.) Phytopath. 30: 15. 1940, and not in 1950 as stated in this Supplement.

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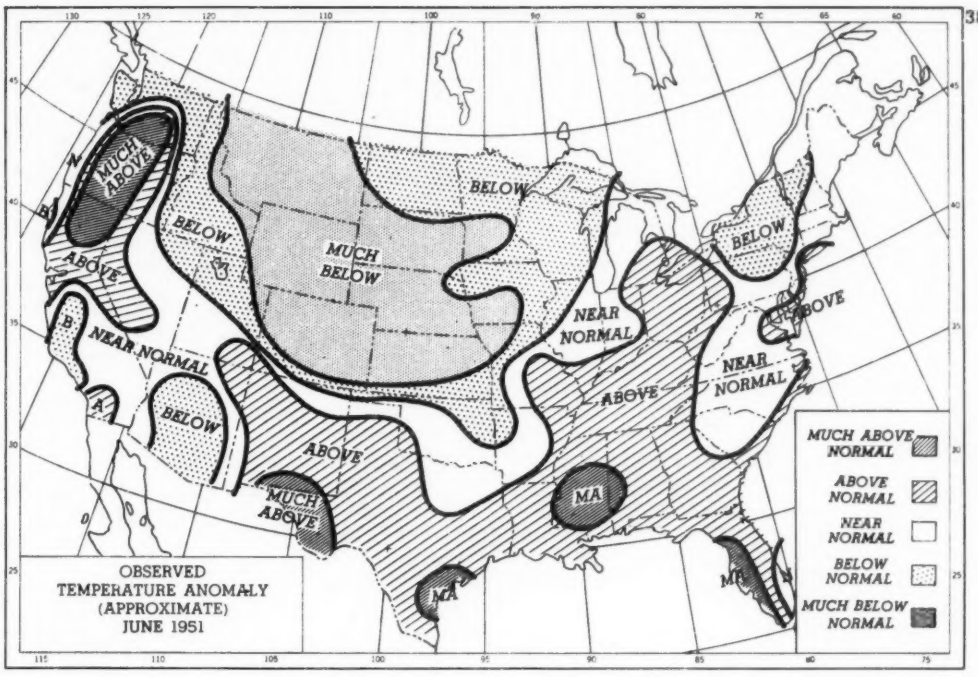
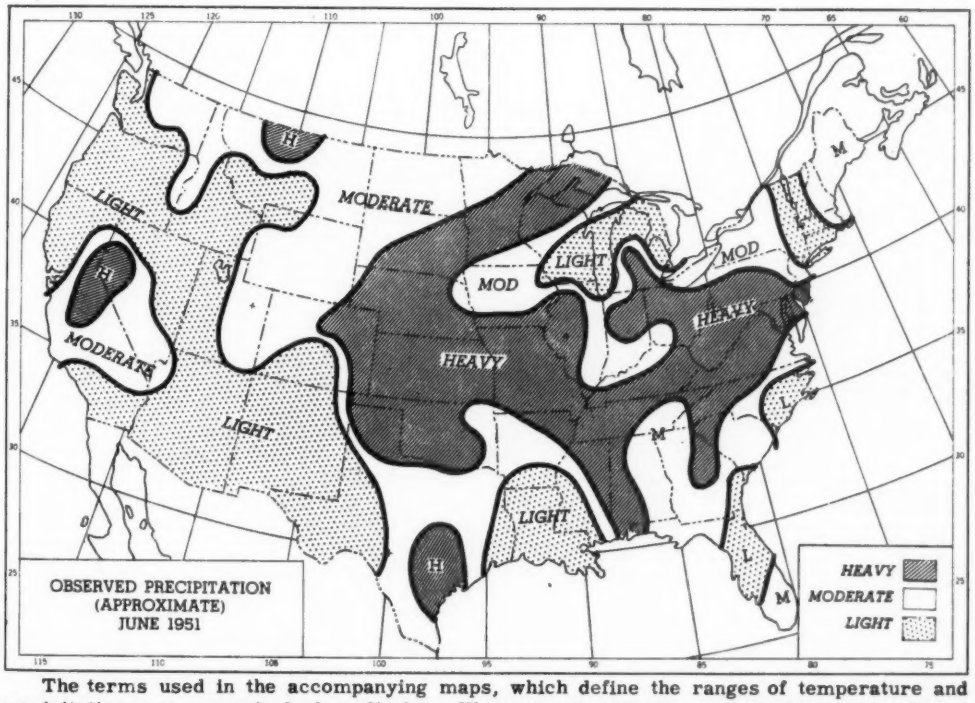
[1]Graham, T. W. Nematode root rot of tobacco and other plants. South Carolina Agr. Exp. Sta. Bull. 390. 25 pp. 1951.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.